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CONTENTS.

—o—

ORIGINAL ARTICLES:—

2

PAGE

n the Origin of the Central Nervous System of Vertebrates. By W. H. Gaskell, M.D., F.R.S. 1

n the Diagnosis of Diseases of the Corpora Quadrigemina. By Prof. H. Nothnagel, M.D. (Vienna) 21

erebral Localisation in its Practical Relations. By David Ferrier, M.D., LL.D., F.R.S. 36

cromegaly. By Prof. Pierre Marie, M.D. (Paris) 59

The Pathology of Sensory Aphasia, with an Analysis of Fifty Cases in which Broca's Centre was not Diseased. By M. Allen Starr, M.D. (New York)... 82

ase of Permanent Conjugate Deviation of the Eyes and Head, e Result of a Lesion limited to the Sixth Nucleus; with Remarks on Associated Lateral Movements of the Eyeballs, and Rotation of the Head and Neck. By A. Hughes Bennett, M.D., and Thomas Savill, M.D. ... ...

Aortic Aneurysm and Insanity. By W. Julius Mickle, F.R.C.P. (Lond.) ... ...

Chorea, with an Account of the Microscopic Appearan Fatal Cases. By H. Handford, M.D., M.R.C.P.

igin of Human Faculty. By George J. Romanes, F

cturnal Paralysis. By Ch. Féré, Medecin de Bicê

Treatment of Certain Diseases of the Spinal

pension. By Motschutkowsky, M.D. (of Od

gical Anatomy of a Case of Tabes Do

sis. By E. St. John Bullen ...

PAGE
The Peroneal Form or Leg-Type of Progressive Muscular Atrophy. By B. Sachs, M.D. (New York) 447
Syringomyelia. By Henry J. Berkley, M.D. (Baltimore) 460
Laryngismus. By William Gay, M.D., M.R.C.P. 4
Katatonie. By W. Julius Mickle, M.D., F.R.C.P. (Lond.) ...
Reflex Spinal Scratching Movements in some Vertebrates. By John Berry Haycraft, M.D. 51
Nuclear Vacuolation in Nerve Cells of Cortex Cerebri. By James R. Whitwell 521

CLINICAL CASES:—

I.—Abscess of the Paracentral Lobule Bursting into the Lateral Ventricle. II.—Aneurism following Embolism of the Anterior Cerebral Rupture into the Right Lateral Ventricle. By Fred. W. Mott, M.D. 14
Chorea Insaniens. By William Gay, M.D., M.R.C.P. 15
Two Fatal Cases of Acute Chorea, with Insanity. By Dr. Evan Powell 15
Asymmetry of the Olivary Bodies of the Medulla Oblongata. By Arthur V. Meigs, M.D. (Philad.) 1
Case of Tumour of the Right Temporo-Sphenoidal Lobe bearing on the Localisation of the Sense of Smell, and on the Interpretation of a Particular Variety of Epilepsy. By J. Hughlings Jackson, M.D., F.R.C.P., LL.D., F.R.S., and Charles E. Beevor, M.D., F.R.C.P. 3
ic Rigidity in a Pedestrian, associated with a Remarkable Neuromuscular Irritability. By W. B. Hadden, M.D., ... 525

GESTS :—

By H. Handford, M.D. 1
Corpus Callosum in the Human Brain, with ew Case. By Alexander Bruce, M.D. (Edin).
llas and Ph. Chaslin (Paris) ... ..

PAGE
Cerebral Localisation in its Practical Relations. By Charles K. Mills, M.D. (Philad.) 233, 358
On Paralysing Vertigo (Gerlier's Disease). By P. Ladame, M.D. (Geneva) 534

REVIEWS AND NOTICES OF BOOKS:—

Guinon: Les Agents Provocateurs de l'Hystérie. By Prof. Marie ... 407
Kerr: Inebriety, its Etiology, Pathology, Treatment and Jurisprudence. By Charles Mercier, M.B. 567
Cullerre: Traité Pratique des Maladies Mentales. By E. Birt ... 570

ABSTRACTS OF BRITISH AND FOREIGN JOURNALS:—

Current Nerve Anatomy and Physiology. By Alex. Hill, M.D. ... 410

UNIVERSITY OF CALCUTTA
LIBRARY
THE ADVANCEMENT OF LEARNING

LIST OF ILLUSTRATIONS.

PAGE
Acromegaly... 61
The Pathology of Sensory Aphasia 83
A Case of Permanent Conjugate Deviation of the Eyes and Head, Figs. 1 and 2 ... ... 104, 108
Chorea, with an account of the Microscopic Appearances in Two Fatal Cases, Figs. 1–7 ... ... 135, 136, 137, 138, 139, 140
Peripheral Neuritis, Figs. 1–33 176
Case of Tumour of the Right Temporo-Sphenoidal Lobe, Figs. 1–6 355, 356, 357
Cerebral Localisation in its Practical Relations, Figs. 1–7 403, 404, 405, 406
Pathological Anatomy of a Case of Tabes Dorsalis, with General Paralysis, Figs. 1–10 440
The Peroneal Form or Leg-Type of Progressive Muscular Atrophy, Figs. 1–5 448
Syringomyelia, Figs. 1–9. Fig. A 464
Nuclear Vacuolation in Nerve Cells of Cortex Cerebri. Figs. A to K 523

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BRAIN.

JULY, 1889.

Original Articles.

ON THE ORIGIN OF THE CENTRAL NERVOUS SYSTEM OF VERTEBRATES.

BY W. H. GASKELL, M.D., F.R.S.

OF the three possible methods—anatomical, physiological, and embryological—of investigating the nature of the invertebrate nervous system from which the vertebrate sprang, the last is in great favour at the present day partly because it is a novelty and partly because it is considered to be a fact that ontogeny reproduces more or less the phylogenetic history. If we glance at the theories which have been held about the origin of the central nervous system we find a distinct difference of view corresponding with the incoming of embryological teaching. The older ideas, based on anatomical and physiological comparisons, before the birth of embryological conceptions, pointed directly to the evolution of the vertebrate nervous system from such invertebrates as the Arthropoda; such comparisons are well exemplified by the writings of Newport in the *Phil. Trans.* of 1834 and 1843, and were defended and extended by Leydig[1] even as lately as 1864.

The newer school of morphologists have disregarded these comparisons and attempted to find the origin of the vertebrate in such forms as the Tunicates, the Nemertines, Balanaglossus, &c. The reason for the change of view has been not only the embryological evidence leading to the transmutation of Vertebrata into a group of the Chordata, but also the difficulty of reconciling the homologies which apparently exist between the different parts of the nervous system in Vertebrata and Arthropoda, with the complete failure of any attempt to discover in the vertebrate any trace of an invertebrate alimentary canal passing between the supra- and infra-œsophageal ganglia.

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[1] F. Leydig., *Bau des thierischen Körpers*, Tübingen. 1864.

Yet it appears to me that the resemblances, both anatomical and physiological, between the central nervous system of Arthropoda and Vertebrata, not only as given by Leydig, but as discovered since, are too strong to be lightly disregarded.

Leydig[1] looks upon the vertebrate brain as corresponding not only to the supra-œsophageal ganglia of the Arthropoda, but to the supra- and infra- œsophageal ganglia taken together, so that the parts of the vertebrate brain correspond to the parts of the invertebrate, if we imagine that the brain was pierced by an œsophagus between the crura cerebri. With this idea as groundwork, he then attempts to follow out the resemblances between the brains of Arthropoda and Vertebrata, in the same manner as, but more fully than, Treviranus, Newport and others had already done, without however attempting to account for the disappearance of the original œsophagus; he comes to the following conclusions.

In insects the brain consists of a number of swellings, all of which are paired; the foremost pair are the lobes from which the antennæ spring—these are equivalent to the lobi olfactorii of vertebrates; the next pair are the main cerebral ganglia, from which the optic lobes can separate as special lobes. The œsophageal commissures are the crura cerebri, and the infra-œsophageal ganglia correspond to the cerebellum and medulla oblongata, from which arise the nerves for the mouth parts.

He then asserts that not only does this resemblance of position exist, but also that Bergmann and Leuckart are wrong in considering the supra-œsophageal ganglia as structurally the same as the lower ganglia of the ventral chain; on the contrary, the structure of the nervous system becomes more complicated as we pass from the ventral chain of ganglia into the infra-œsophageal and supra-œsophageal ganglia, and in the latter especial structural peculiarities exist, so that a structural difference exists between the brain and ventral chain of the Arthropoda, just as between the brain and spinal cord of vertebrates.

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[1] *Op. cit.* p. 185.

Further[1] he points out that physiological as well as anatomical resemblances have been shown to exist; thus he quotes Faivre, who, working on Dytiscus, says, if the brain lobes are removed the animal can walk and swim, but the will to move and the power of regulating the direction are gone. If further the infra-œsophageal ganglia be removed as well, then the animal can still move all its legs but cannot co-ordinate or regulate its movements, so that it can no longer carry out any proper walking or swimming movements.

According then to Leydig in 1864, the brain of the arthropod corresponds to that of the vertebrate in that they both consist of three separate parts which correspond in anatomical position and in physiological significance. They both possess special paired ganglion masses, in the absence of which the will to execute movements is lost. These might in both cases be called the cerebrum or fore-brain. They both possess special paired ganglion masses connected with vision; these might in both cases be called the optic lobes, if we include in that term the optic thalami, and speaking generally, this part might be called the mid-brain. They both possess special nerve structures, in the absence of which co-ordination of movements for such acts as walking or swimming is no longer possible; these might in both cases be called the hind-brain.

Since this time evidence has been accumulating to confirm and strengthen the position taken up by Treviranus, Newport, Leydig and others. Thus to give one or two examples, I would refer to Bellonci's[2] paper on the brain of *Spharoma serratum*, where he describes the supra-œsophageal ganglia as consisting of a superior segment, a

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[1] *Op. cit.* p. 187.

[2] 'Archiv. Ital. de Biologie,' vol. i., p. 176. (See Fig. 1, Pl. 1.)

middle segment, and an inferior segment. The superior segment constitutes the cerebrum and may be looked upon as the cerebral hemispheres or fore-brain; the middle segment gives origin to the optic nerves and to the olfactory nerves, and may be looked upon as the mid-brain and optic thalami; the inferior segment is not strictly speaking supra-œsophageal, but is situated on the œsophageal commissures and consists especially of a ganglion on each commissure which is the stomatogastric ganglion and gives origin to the nerves of the alimentary canal; in addition the nerves of the external antennæ arise from this region. Upon the assumption that the crura cerebri represent the œsophageal commissures, then, as I have pointed out in my last paper, we may perhaps compare the stomato-gastric ganglion with the cells of the *substantia nigra*, and may look upon the infra-œsophageal ganglia, together with this inferior segment of Bellonci, as constituting the hind-brain.

We see then that not only the more ancient but also the more modern researches indicate an anatomical separation of the brain of a crustacean into three parts, which correspond in relative position to the fore-brain, mid-brain with optic thalami, and hind-brain of the vertebrata.

From the physiological side the evidence is still stronger; if we take first the fore-brain or cerebral hemispheres we may use the phraseology of Hughlings Jackson and speak of them as connected with the spinal cord nervous system by a well-defined system of nerve tracts or commissures which are extrinsic to the level of the spinal cord system; now one of the most striking peculiarities of this higher level system is its inhibitory power over the activity of the lower centres.

Taking next the second division, *i.e.*, the optic lobes with the optic thalamus, we see clearly that here is the centre for the nerves of vision, and that also in the past history of the vertebrate there existed in connection with that part of the optic thalamus known as the ganglion habenulæ an extra visual organ of an invertebrate type, viz., the pineal eye; also if Hill[1] is right the ultimate origin of the olfactory nerves is to be found in the optic thalamus.

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[1] Hill 'Plan of the Central Nervous System,' Cambridge, 1885.

In the third place the cerebellum with the pons varolii and the semi-circular canals are recognised universally as that part of the brain which is especially concerned in the maintenance of the equilibration of the body and in the co-ordination of such complicated movements as walking, swimming, &c.

Evidence as to the seat of the corresponding functions in the brain of Arthropods is given in a most able paper by Ward[1] upon the brain of the crayfish, in which he shows that section of the œsophageal commissures not only removes the power of the will over the movements of the animal, but also removes powerful inhibitory influences which normally pass down from the supra-œsophageal ganglia to the lower centres, as is evidenced by the unceasing rhythmical movement of the appendages after separation of the supra-œsophageal ganglia. Such influences may pass from the superior segment of Bellonci, which I have compared to the cerebral hemispheres, or from the middle segment which corresponds to the optic lobes and optic thalami. At present we cannot separate these two with respect to their inhibitory power. Physiologically, then, the superior segment of the supra-œsophageal ganglion corresponds to the cerebral hemispheres, especially if we take into account at the same time the function of the closely-connected optic part of the brain. So also the middle segment, which gives rise to the optic and olfactory nerves, clearly corresponds to the optic lobes and optic thalami as far as function is concerned.

Ward's experiments also confirm Leydig's view as to the functions of the sub-œsophageal ganglia; he concludes that the second or sub-œsophageal ganglia have functions so nearly equal in importance to the supra-œsophageal as to justify Leydig's comparison of the two together with the brain of a vertebrate pierced by an œsophagus between the crura cerebri, and he proves that the sub-œsophageal ganglia are the centres *par excellence* for co-ordinating the movements of the limbs. We see then that Bellonci's inferior segment together with the sub-œsophageal ganglia, correspond physiologically to the region of the cerebellum and pons varolii;

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[1] Ward *Journ. of Physiol.*, vol. ii.

nor is this all, for so close is the parellelism between the two that even the curious part played by the semi-circular canals in the maintenance of the equilibrium of the body finds its counterpart in the functions of the otocysts of the crustaceans. This observation has been made by Delage[1] who has found that the removal of the otocysts at the base of the antennæ in various decapods is followed by distinct disturbances of equilibration.

Delage goes so far as to draw a direct comparison between the otocyst and the membranous labyrinth of the vertebrate ear. He says,[2] "The otocysts represent in a stage of imperfect development, the membranous labyrinth of the vertebrates. We see in fact that in the latter the semi-circular canals and cochlea exist only in the higher classes; as we descend in the scale we see that the cochlea becomes simpler and simpler and finally disappears, while the semi-circular canals are greatly reduced in the lowest fishes (Myxine). It is therefore clear that in spite of its complication the membranous labyrinth of the vertebrates is derived from a single vesicle with smooth walls, in all respects similar to an otocyst; a further proof of this is given by the evidence of the ontogenetic development of that organ."

He then goes on to say in what way he imagines that the double function of the auditory organ may have arisen phylogenetically. "The function of the simple auditory vesicle of the primitive vertebrate, like the otocyst of the invertebrate, must have been for the purpose of perceiving noises (bruits), and of regulating locomotion. It would then become separated into two parts, each of which was told off for one of these functions, the saccule for the first, the utricle for the second. Then little by little diverticula would be developed from the central parts: the cochlea to perceive sounds (sons), with their qualities of pitch (hauteur) and timbre, no longer mere noises differing from each other only in intensity; and the semi-circular canals, perhaps for the purpose of co-ordinating the movements of the eyes with those of the head, so as to get rid of the visual illusions, which are produced upon movements of the head when the eyes are immoveable."[1]

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[1] Delage, "Archiv. de Zoolog. experiment. et generale," vol. v. 1887.

[2] *Op. cit.* p. 22.

So far we may sum up as follows: the nervous systems of the Crustacea and Vertebrata resemble each other in the following particulars. In both cases the fore part of the brain forms a distinct cerebrum, removal of which removes the will power of the animal and makes it a more or less elaborate reflex machine. In both cases this cerebrum has special inhibitory power over the lower centres. In both cases a special part of the brain, in close connection with the cerebrum, is set apart for the origin of the nerves of sight and smell. In both cases this fore-part of the brain is connected with the hind-part of the brain by means of commissural tracts known respectively as the œsophageal commissures and the crura cerebri. In both cases the hind-part of the brain is concerned in the maintenance of the equilibrium of the animal. In both cases the auditory capsule contains not only the organ of hearing but special apparatus subservient to the maintenance of the equilibrium of the animal.

Further if we consider those nervous elements which we have classed together as being of the same level as the elements of the spinal cord, we find in the vertebrate, as I have pointed out previously,[2] that we are dealing with nerve structures which form the origin of a series of segmental nerves, and also the central inter-segmental communications of such neuromeres. I will not here give again in detail the arguments which I have already used, to show how the segmental cranial nerves are built up on the same plan as the spinal, how the nerve cell groups in the two regions correspond in position and function, but will refer my reader to my two previous papers.[3] Here I will only state the conclusion to which I have arrived, which is the same as has been taught by Schwalbe and others for many a long day—viz., the spinal cord, and its continuation cranialwards, constitutes a well-defined nervous system which is formed by a bilateral series of ganglia, connected together transversely and intersegmentally, which give rise to a series of segmental nerves, all of which are built up on the same plan; the whole forms a uniform system which is connected with the system of the brain, already described, by a number of well-defined commissural tracts, described by Hughlings Jackson as being extrinsic to the level of the spinal cord.

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[1] *Op. cit.* p. 23.

[2] *Journ. of Physiol.*, vol. 10. p. 153.

[3] *Journ. of Physiol.*, vols. 7 and 10.

Here then we see a direct parallelism in both anatomical arrangement and subordination of function to higher centres, between the spinal cord of vertebrates and the ventral chain of ganglia, in the crustacean with their transverse and intersegmental connections, and their connecting commissural fibres with the cerebral nervous system.

The parallelism, according to Leydig, is still closer, for he believes that the segmental nerves of the crustacean show a division at their origin into two parts, corresponding to the anterior and posterior roots of the vertebrate.

We see then that no difficulty exists in the comparison between the nervous matter of the crustacean and that of the vertebrate central nervous system, both in its anatomical and physiological relations.

Indeed the consideration of the phylogenetic development of the nervous system in the Vertebrata enables us to compare in still more minute detail than in the sketch I have just given the nervous system of the two classes; at the end of this paper I will point out how the origin of the vertebrate nervous system from one of a crustacean type not only gives an explanation of a number of obscure anatomical appearances in the brain of the Vertebrata, but at the same time helps to fix homologous parts in the two nervous systems.

I conclude then that there is no difficulty in tracing up the parts of the most highly developed central nervous system from a system of an arthropodan type. This would doubtless have been long since accomplished as the continuation of Leydig's work, but for the difficulty of the invertebrate alimentary canal.

The attempts which have been made by Dohrn and others to locate the original mouth in the neighbourhood of the fourth ventricle, and then to pass the œsophagus through the infundibulum by way of the pituitary body into the alimentary canal, and so to make the vertebrate derived from an invertebrate ancestor, whose dorsal surface has become ventral, have conspicuously failed, and have indeed been given up by Dohrn himself. With this failure it has been supposed to be hopeless to pierce the anterior part of the vertebrate central nervous system with an œsophageal canal, and in consequence the comparison of the nervous system of the vertebrate with that of the arthropod has been given up.

In my paper published in the *Journal of Physiology*, vol. x., and in a paper which I read before the Neurological Society in the summer of 1888, I have explained how in my opinion the remains of the old invertebrate alimentary tract is still existent in the vertebrate nervous system. I will not again here repeat what I have already published, but will briefly indicate the nature of the arguments used and the conclusions arrived at.

We find throughout evidence of a non-nervous tube, which is mixed up with the formation of the nervous system proper.

This non-nervous tube is the remains of the old alimentary canal, and was of the type of the crustacean canal, with a large cephalic stomach, and a straight, simple intestine opening into an anus. The straight, simple intestine forms the canal of the spinal cord, and its walls have become modified to form the supporting tissue or myelo-spongium of the nervous elements of the cord; it passes free from admixture with nervous elements, as the neurenteric canal of the embryo, into the anus.

The remains of the non-nervous cephalic stomach are well seen in the cephalic region of the nervous system in the shape of the non-nervous epithelial structures, which are so freely found there, as part of the walls of the central tube, and which, by being thrown into folds, form on the dorsal side the choroid plexuses, and on the ventral side the saccus vasculosus. The remains of the mouth and œsophagus are found as a folded-down tube, which passes from the third ventricle, forms the infundibulum with the lobi infundibuli, then remaining dilated and epithelial in character, forms in the fishes the saccus vasculosus, and finally is bent down on to the surface of the brain to near the exit of the third nerves, being occluded by the compression of its walls, and by a degenerative modification of the cells of its terminal portion. The pituitary body is situated on the anterior lip of this tube against the dilated portion known as the saccus vasculosus. In this way not only does the arrangement of the nervous material in the two classes remain the same, but an explanation is given of the non-nervous structures found in connection with the nervous tube of the Vertebrata.

By this explanation of the relation between the non-nervous and nervous material, the crustacean merges insensibly into the vertebrate without any shifting of dorsal and ventral surfaces; the supra- and infra- œsophageal ganglia remain the same in position and in function; the œsophageal commissures remain; mouth, œsophagus, intestine, anus, are all there in the same position, with respect to the nervous elements, as in the crustacean ancestor.

Hitherto no explanation has been given of the reason for the non-nervous epithelial structures in the brain of the vertebrate: in the pre-Darwinian days it was possible to say that these epithelial parts of the roof of the nervous tube in the cranial region were potential nervous matter, were for the purpose of enabling the development of the brain to take place as the vertebrate rose in the scale of creation; now however such an explanation is recognised as unmeaning and yet no other is given in its place. We cannot say that it is nervous material which has thinned down and degenerated, for all the evidence of comparative anatomy goes to show that the lower the animal in the scale of evolution, the more conspicuous is this epithelial bag, and the less is it obscured by the growth of nervous matter. In fact we are driven to conclude that the epithelial bag is pre-existent and that the nervous matter is situated in definite places on the outside of it, as I have already said.

Again, it is significant in connection with the comparison between the nervous system of the vertebrate and the combined nervous and alimentary systems of the crustacean, to find that the cephalic stomach of the crustacean is a non-glandular, simple epithelial bag, for the purpose of holding food and not for digesting it; in this respect also the non-nervous membranous structures of the vertebrate brain, with their well-known simple epithelial character, agree with the theory which I have put forward. So also the simple, straight intestine of the crustacean is free from any digestive glandular structures, and is simply either an excretory duct, or perhaps absorbent in function.

In the crustacean however in addition to the intestine and stomach, and in connection with the latter, there exists a large and most important organ, the so-called liver, and it is in the cells of this gland that the digestive ferments are formed, and by means of its secretion the food taken into the stomach is rendered capable of absorption, either by the intestine, or perhaps partly by the liver itself. It is a large, symmetrically bilateral gland, spreading over the whole of the cephalic region; its ducts enter into the alimentary canal at various places, or else combine as a single duct to enter the stomach near its pyloric end on the ventral surface.

Seeing then the large size and great importance of this organ in the crustacean, it follows that if the theory set forth in these pages is a true one then some vestige of this important adjunct of the crustacean alimentary canal ought most certainly to be found in connection with the canal of the vertebrate nervous system in the cranial region.

A comparison of the cranial cavity of the lowest and highest vertebrates brings prominently forward the great difference in the extent of space occupied by the brain mass at the two extremities of the vertebrate kingdom: on the one hand in man the nervous matter so closely packed within the cranium as to leave its impress upon the bony walls of the cavity; on the other hand in the fishes a brain so small lying in a cranial cavity so much out of proportion that the greater part of the cavity is unoccupied by nervous matter. Here again, just as in the case of the membranous parts of the brain tube, the explanation which might have been given, and indeed was given in pre-Darwinian days can no longer be accepted now—the explanation, viz., that in the lowest vertebrates a small brain was designedly contained in a large cavity in order to allow room for the increase of the brain material as the animal ascended in the scale of creation; yet no other is put in its place, no explanation as far as I know is given why the large space between brain and brain case in the lowest vertebrates is occupied by a mass of peculiar jelly-like tissue, while in the highest vertebrates nothing of the sort is to be found. The reasonable argument appears to me to be not that this mass of gelatinous-looking tissue has been formed for the purpose of supporting and steadying a brain which is too small for its case, but rather that it represents some pre-existing organ which, together with the brain, filled up the cranial cavity, and that having lost its original function it has become converted into a mass of soft jelly-like material. The pre-existing organ is in my opinion the so-called cephalic liver of the crustacean ancestor. My reasons for this opinion are based on the study of this structure in the ammocœte and I am at present engaged in writing out a paper on the brain of the ammocœte in which I intend to discuss fully the nature of the evidence which has led me to this conclusion. It will be sufficient here to say that this tissue is composed of large closely packed glandular-looking cells which are arranged so as to form a distinct organ symmetrically placed on each side of the middle line of the brain; this organ extends as a compact mass in front of the cerebral hemispheres, and extends in the shape of scattered cells into the region of the spinal cord. At one place this tissue forms an apparent hilus and here in the nervous matter the remains of a tube passing from the commencement of the fourth ventricle to the surface are to be found; this spot is the region of the *ganglion interpedunculare* or the *conus post-commissuralis* (Fritsch). That this *conus post-commissuralis* contains within itself the remains of a diverticulum from the central cavity of the nervous system is recognised by Ahlborn[1] and in confirmation of the view that it represents the rudiment of the duct of the original cephalic liver of the crustacean ancestor we find that it opens into the central canal at the lower limit of the fourth ventricle—*i.e.*, according to the theory here put forward, into the pyloric end of the original stomach; that it comes to the surface of the brain on the ventral side at the spot where the gelatinous tissue converges to form a hilus, just as in the crustacean ancestor the original liver duct must have entered into the so-called liver at this point.

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[1] Ahlborn. *Zeitschrift f. Wissent. Zoolog.*, vol. xxxix., 1883.

In this way then it seems to me do we obtain a good and sufficient reason in accordance with modern evolutionary ideas, for the apparent emptiness of the brain case in the lowest vertebrates; the large space in question was filled by that large and important organ of digestion, the so-called liver, which occupies so great a space in the crustacean economy. With the loss of function of the original alimentary canal and the increasing growth of the nervous system, this organ also lost its function and its cells became converted by that curious mucilaginous degeneration into the so-called arachnoidal fat tissue which is not real fat tissue, and is therefore called by Ahlborn "*Arachnoidale Füll-gewebe.*"[1] After it had lost its original function this tissue still remained well-supplied with blood vessels and according to Sagemehl[2] perhaps took on the function of lymph tissue; with the increase and growth of the brain it was gradually compressed out of existence altogether until at last in the human brain the cranial cavity is filled with nervous matter, and the remains of the old original cephalic liver are perhaps to be seen in the so-called *Glandulæ Pacchioni.*

We see then that by this theory of mine a perfectly straightforward and reasonable explanation is given of two hitherto insoluble anatomical problems, the one the reason for the existence of the choroid plexuses, and the other the reason why the cranial cavity in the lower vertebrates is so largely filled up with a non-nervous gelatinous semi-fluid mass of tissue. The truth of a theory however depends not only upon its being able to explain one or two striking difficulties but even in small details its correctness ought to appear. This appears to me to be the case. In the brain of the vertebrate or in connection with it, are many peculiar structures, the function of which is unknown and which are apparently the remains of originally important structures, for they are found with great constancy throughout the vertebrate kingdom. Such are the hypophysis and epiphysis, the ganglia habenulæ and Meynert's bundle, the tænia thalami, the ganglion interpedunculare and the substantia nigra. Of these there is no evidence that the hypophysis is of the nature of a sense organ; on the contrary, its glandular character is manifested both by histological and pathological investigations. It is in some animals, according to Dohrn, a paired organ; there is no evidence that it communicates with the central canal of the nervous system. It is in all probability the remains of a primitive glandular structure, and its interpretation is so closely bound up with the question whether or no it is a paired organ, that it is better to wait until that question is settled before speaking with any certainty about its homologies.

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[1] F. Ahlborn, *loc. cit.* p. 287.

[2] *Morphol, Jahrbuch.* vol. ix. p 457.

The epiphysis is clearly, as the result of recent investigations shows, the rudiment of a median eye; such eye is usually spoken of as having been vesicular, and it has been suggested to me that it is difficult to obtain a vesicular eye from a crustacean ancestor. In my paper on the ammocœte brain I propose to give the appearances presented by the eye in the young lamprey and to show that its structure is not vesicular, but closely resembles the median eye of Limulus as described by Ray Lankester.[1] In fact the position of this pineal eye, its relation to the *ganglia habenulæ* and its structure, all fit in well with the supposition that it represents the median eye of a crustacean ancestor of an antique type, such as is represented by the king-crab.

With respect to the *substantia nigra*, I have already suggested that it may represent the primitive stomato-gastric ganglia, partly from its position on the crura cerebri, partly from the deeply pigmented character of its cells, which points directly to a loss of function, and partly from its apparent connection with the degenerated tissue in the roots of the oculomotor nerve. The rest of the structures which I have mentioned at the beginning of this section, viz., the ganglia habenulæ, tænia thalami, Meynert's bundle and ganglion interpedunculare, form a group of considerable interest from the point of view of this paper; for the lower we descend in the scale of evolution of the vertebrate the more do these structures become prominent, the more evident is it that they represent distinctly very primitive parts of the nervous system.

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[1] *Quarterly Journ. Microp. Science*, vol. xxv.

We see that the phylogenetic development of the nervous system in vertebrates largely consists in the great increase of those parts which correspond to the supra-œsophageal ganglia and their connections with the subordinate system of the spinal cord. Thus the superior segment of Bellonci, viz., the cerebral hemispheres, increases enormously in size and complexity.

The optic thalami which overshadow and include the ganglia habenulæ in the higher vertebrates, are in the ammocœte but small in comparison with the conspicuous and important ganglia habenulæ.

The optic lobes vary in size according to the perfection of the visual apparatus, and in the ammocœte, before transformation takes place, they can hardly be said to exist, although at this time both the pineal eye and the ganglia habenulæ are well developed. In the primitive crustacean-like ancestor of the vertebrates, then, it appears to me that the superior segment of the supra-œsophageal ganglia was represented by the cerebral lobes, while the middle segment was represented essentially by the ganglia habenulæ and the optic thalami. It follows then, that the *tænia thalami*, which according to Ahlborn are well represented in the ammocœte and form an extensive system of peculiarly coloured fibres which pass symmetrically on each side from the *tubercula intermedia* (ganglia habenulæ) into the cerebral hemispheres, represent the original connection between the superior and middle segments, while the Meynert's bundles represent the connections between the middle segment and some part of the sub-œsophageal ganglia.

Whether Meynert's bundles are connected with the ganglion interpedunculare or not Ahlborn was unable to assert definitely; they come to the ventral surface of the brain near that region but their ultimate destination is not yet settled.

If I understand rightly the part of the brain recognised by Ahlborn as the ganglion interpedunculare, then this so-called ganglion represents in my opinion the occluded termination of the duct of the cephalic liver, as I have already said; the details of its structure in the ammocœte upon which this opinion is based will be given in the paper already alluded to.

Further in considering the hind-brain it is very instructive as already pointed out in my former paper, to observe the growth of the cerebellum, so large and important in the highest vertebrates, so small and insignificant in the ammocœte that Ahlborn could only recognise it with difficulty. In its inception it must be looked upon, as Osborn[1] has shown, as a band of nervous tissue stretching over the dorsal side of the neural tube, in close contiguity to the fourth nerve. Then before the cerebellar hemispheres are formed, we find this band of nervous material surrounding a loop of the membranous roof, and so forming the worm of the cerebellum. By this solidification of the walls of the pinched-up loop it follows that two lateral bags of the membranous roof are formed; on the external surface of the floor of these two bags two ear-shaped ridges of nervous tissue are found, called the *fimbriæ*, which ultimately by further growth form the cerebellar hemispheres, as I have explained in my last paper.[2]

We see then that the evolution of the cerebellum not only shows in a most instructive manner how the enlargement of the brain is formed by the steady growth of nervous material over the original non-nervous epithelial tube, but also points directly to the conclusion that in the primitive condition the nervous material which corresponded in function with the cerebellum, and from which by gradual increase of growth the cerebellum has been evolved, was situated on the ventral and not on the dorsal side of the original neural tube. In other words the cerebellum and semi-circular canals are both concerned in the function of equilibration, just as are the sub-œsophageal ganglia and the otocysts, because in each case they are the direct lineal descendants of these parts of the crustacean nervous system.

[1] *Journ. of Morphol.*, vol. ii. [2] *Jour. of Physiol.*, vol. x., p. 194.

Again, in the spinal cord it is easy to see how the evolution of the nervous system has brought about the marked separation between the exits of the anterior and posterior roots, which is so marked a characteristic of the cord of the higher vertebrates; for we see that with the growth of the cerebral hemispheres and cerebellum we must have an increasing growth of the long commissural tracts which connect the lower centres of the spinal cord, or ventral chain, with these two great masses of brain matter. This means essentially an increase in the pyramidal and cerebellar tracts, and it is clear that if the crossed pyramidal tract and the direct cerebellar tract be either removed or very much diminished, then the anterior and posterior horns, with the exits of the corresponding roots, must approximate to each other more and more closely.

Finally, this theory explains, as I have already pointed out in my former paper, what has always been hitherto without explanation, viz., the meaning of the formation of the cerebral vesicles in the embryo. If we keep steadily in view the conception that the nervous system is formed around and on the outside of a pre-existing non-nervous epithelial tube, the anterior dilated part of which was the original cephalic stomach, then we see how the evidence of ontogeny confirms this conception and how far it indicates the line of phylogenetic descent. At first the neural tube is formed with a simple dilated anterior extremity, indicating its origin from a simple intestinal tube with a dilated anterior cephalic stomach. Then as it continues to grow into a vesicular form it ceases to dilate uniformly, a constriction appearing on its dorsal surface at one particular place so as to divide it into an anterior and posterior vesicle; this constriction denotes the growth of the nervous matter of the ganglia habenulæ and posterior commissure and indicates the position of the optic portion of the supra-œsophageal ganglia upon the original cephalic stomach.

The posterior vesicle now becomes divided into two portions by a constriction which again corresponds to the formation of nervous matter upon the non-nervous tube, viz., the fourth nerve and commencing cerebellum. By this means the brain tube is divided into the three cerebral vesicles, the dorsal walls of which are membranous except at the place of the constrictions.

Then again the first cerebral vesicle becomes divided into two to form the fore-brain and thalamencephalon, while the third cerebral vesicle is also divided into two to form the cerebellum and medulla oblongata.

In this way then we see the formation of five cerebral vesicles, viz., prosencephalon, thalamencephalon, mesencephalon, cerebellum and medulla oblongata, and it is clear, as Osborn[1] has pointed out, that the divisions between these vesicles are formed by a series of commissural bands of nervous matter. Of these the limiting nervous strands between the thalamencephalon and mesencephalon, and between the mesencephalon and the hind-brain are of primary importance; while the separation into prosencephalon and thalamencephalon, and into cerebellum and medulla oblongata, are of subordinate importance.

Not only the posterior commissure and ganglia habenulæ, but also Meynert's bundle are most constant throughout the vertebrate kingdom, and it is instructive to note how prominently these latter fibres stand out in sections of such low forms as Petromyzon in its young state. I suggest therefore that the primary constriction which separates the thalamencephalon from the mesencephalon is an indication of the formation of the ganglia habenulæ, posterior commissure and Meynert's bundle, *i.e.*, of the middle segment of the supra-œsophageal ganglia with its connecting tract with the subœsophageal ganglia. There is nothing new in the suggestion that Meynert's bundle on each side is in the position of the lateral constriction which separates the thalamencephalon from the mesencephalon; it has been already made by Paul Mayser, and in Ahlborn's paper[2] a discussion of Mayser's view is given, to which I must refer my reader.

Again, the next most important constriction is the place where the valvula cerebelli is found in the adult and indicates the separation between mid-brain and hind-brain; here we find again in all vertebrates the crossing over of a nerve tract from the ventral to the dorsal side, viz., the fourth nerve; and in close connection with this is the original commissural band of fibres which constitutes, according to Osborn, the rudiment of the cerebellum. The meaning of this crossing over of the fourth nerve and of the formation of these bands of nervous material in this region I must leave until the discussion of the meaning of the cranial nerves as a whole, in connection with the origin of vertebrates from a crustacean-like ancestor.

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[1] *Op. cit.* [2] *Op. cit.*, p. 235.

With respect to the other two divisions, the separation between cerebellum and medulla oblongata is clearly an indication of the hinder limit of the formation of the cerebellum itself, and depends therefore for its distinctness mainly upon the extent to which the cerebellum is developed in the adult condition. Thus in the lamprey Ahlborn[1] was unable to satisfy himself of the existence of this constriction.

The division of the fore-brain into prosencephalon and thalamencephalon is recognised now as a constant and important division, but it does not appear to be absolutely settled what the dorsal constriction corresponds to in the adult state. The most generally held view is that the anterior commissure marks the position of the dorsal limit between these two parts of the brain. Osborn however considers that his superior commissure is the true limit, and not the anterior.

The difference comes to this: on the first view the prosencephalon forms the cavity of the cerebral hemispheres and the nervous matter overlying them, while the thalamencephalon forms the cavity of the third ventricle with its membranous roof which forms the choroid plexus of the third and lateral ventricles and its lateral nerve masses the optic thalami together with the pineal gland. According to Osborn's view, seeing that his superior commissure separates the supra-plexus (*i.e.*, the choroid plexus of the third and lateral ventricles) from the pineal gland, it follows that his prosencephalon includes this choroid plexus as well as the cerebral hemispheres, while his thalamencephalon consists on the dorsal side only of what is included between the superior and posterior commissures, *i.e.*, the pineal body, with I presume the optic thalami forming its lateral walls.

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[1] *Op. cit.*

Of these two views it seems more reasonable to look upon the supra-plexus (choroid plexus of third and lateral ventricles) as belonging to the thalamencephalon, so that the anterior commissure would in this case represent the limit of the first vesicle or prosencephalon. This commissure is said to be very constant among vertebrates; it forms a connection between the two cerebral hemispheres, and is said to be the first transverse commissure of the cerebrum (Quain) which is developed in the embryo. It represents probably the intercommunication between the two lobes of Bellonci's superior segment, and forming the posterior boundary of the cerebral hemispheres causes a well-defined constriction in consequence of the growth of the cerebrum forwards.

The increasing study of this question convinces me more and more that the view herein expressed is right. I have however thought it best to bring out my investigations bit by bit as they are ready for publication, rather than to wait some years and then publish the results of my investigations as a whole. I propose then to publish a series of papers in support of the view here expressed, dealing with the question of the origin of vertebrates from the crustacean from every point of view, both anatomical and palæontological. I reserve to myself the right of following up this investigation and propose after the publication of my paper on the central nervous system of the ammocœte to deal with the meaning of the cranial nerves, and therefore with the formation of the present vertebrate alimentary canal; for as I have pointed out in my last paper it is clear that the hindmost group of cranial nerves which arise from the medulla oblongata possess peculiarities of origin and distribution which are due to the formation of the present alimentary canal, just as the peculiarities of the foremost group of cranial nerves are due to the loss of function of the old alimentary canal and the parts connected with it.

Cambridge, June 1st, 1889.

ON THE DIAGNOSIS OF DISEASES OF THE CORPORA QUADRIGEMINA.

BY H. NOTHNAGEL (VIENNA).

WHEN I, ten years ago, formulated on the basis of the then existing clinical material some diagnostic propositions relating to diseases of the corpora quadrigemina, I was constrained to submit them with great reserve. The eventual accuracy and value of those data for establishing the diagnosis required further clinical testing. Bernhardt[1] soon afterwards expressed himself just as cautiously; he left the question open, whether it be possible to diagnose tumours of the pineal gland and corpora quadrigemina.

In my first publication,[2] disregarding the scanty clinical records to be found in literature, I treated of a striking case observed by myself. Bernhardt two years later collected eleven cases of tumour of the corpora quadrigemina. Other cases have since been published, and I have met with three more cases of these rare tumours, which were investigated clinically and by subsequent dissection. In two of them I made the correct diagnosis during life. This might be regarded as an accident; nevertheless, the fact that it is possible to form a correct diagnosis of disease in the quadrigeminal region always convinced me that the grouping of the clinical phenomena which led me to the diagnosis must have a significance. More extended observations will prove or refute the correctness of my opinion.

Three[3] of my own cases already have been published; the clinical history of the fourth is related below. I abstain

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[1] 'Contributions to the Symptomatology and Diagnosis of Cerebral Tumours,' Berlin, 1881.

[2] 'Localisation of Diseases of the Brain,' Berlin, 1879.

[3] *Ibid.* p. 206. *Wiener med. Blätter*, 1882 and 1888.

016238

from adding a fifth case (in which I diagnosed disease of the corpora quadrigemina during life, which was verified by the autopsy), because I only saw the patient once in consultation practice, and possess no notes thereof.

Of course it would be desirable to base conclusions upon old stationary focal lesions (hæmorrhage and softening) if sufficient material were available; but such lesions very seldom occur in the quadrigeminal region, and still more rarely (so that scarcely an usable case thereof is recorded in the literature) is a hæmorrhage or softening limited to the corpora quadrigemina alone. We must therefore seek to decide whether adequate grounds for the diagnosis can be obtained from the cases of tumours.

I omit a reproduction of the current views regarding the functions of the quadrigeminal bodies, only with reference thereto it must be remembered that no harmony exists. Bechterew especially has recently by no means corroborated most of the earlier teaching, and on the ground of his experiments he restricts the relationships of the corpora quadrigemina to the visual sense alone. We therefore are so much the more exclusively directed to critical estimation of the clinical material, unmoved therefrom whether our results accord with present physiological views or not.

Joseph E., æt. fifteen years, without hereditary taint, formerly always healthy. Three years before his reception into hospital he fell from a tree, striking the ground first with his feet, then with his head, and for a short time was unconscious. Vomiting did not occur. He remained fourteen days in bed on account of a wound of his foot. The first symptom, which appeared after the lapse of some time (the exact time of its commencement could not be determined), was an unsteadiness of gait, which never disappeared—on the contrary has grown worse. Patient staggered constantly, went zig-zag and often fell to the ground. In the winter of 1886-7 violent pains in the head and eyes developed, accompanied by nausea. At that period vomiting recurred several times nearly every day, and patient noticed a discharge from his right ear, which lasted a month, then ceased. In the summer of 1887 his condition improved, but next winter severe cephalalgia again set in, at first without emesis, later with frequent nausea often ending in vomiting, which has never completely disappeared. From the early part of 1888 increased feeling of dizziness on walking and more frequent tumbles were complained of. Since the year 1886 progressive disturbance of vision developed, so that the patient became able to read none but large type. Rectal and bladder troubles were absent. Appetite good.

Present State, Aug. 21st, 1888.—Patient is strongly built for his age, well nourished, assumes an easy dorsal posture. The sensorium (Das Sensorium) is intact; no headache. Head is remarkably large and broad; its circumference over the occipital protuberance and frontal eminences is 56 cm. Percussion of cranium not painful. Complexion is fresh and ruddy. Both eyeballs are rather prominent; there is slight convergent strabismus: movement of the eyes, especially of the left, is much limited in outward and upward directions. Pupils equal; react very slowly. The ophthalmoscope shews bilateral neuritis with commencing choked disc. Audition is impaired in both ears; oliatric examination discloses a chronic catarrh of the middle ear; whether simultaneous affection of the sound-perceiving apparatus exists cannot be ascertained. The remaining cranial nerves are unaffected. Pulse 64; the arterial tube is normal, well filled; pulse wave and pulse tension are of medium height. Respirations 20. Temperature 36.4. Neck, short; chest well formed, shews normal respiratory movements. Percussion note of lungs is normal; respiratory sound everywhere is purely vesicular. Cardiac dulness not enlarged: impulse normal in position and characters; heart-sounds pure, over all the orifices. Abdomen somewhat below the level of the thorax soft, tympanitic. Hepatic and splenic dulness not increased.

The upper extremities are moved freely; their muscularity is strongly developed: compressive power of hands is equal and of medium force. Myotatic irritability is normal. Triceps tendon reflex not demonstrable. The musculature of the legs is equally well developed; their movements in bed are not impaired: patient raises the extended limb in spite of strong opposing pressure on the knee. Gait is wide-based, unsteady, and reeling—all parts of the lower limbs being freely movable, save that the inner border of the right foot is drawn up. The staggering occurs especially during slow locomotion; the brisk walk is certainly unsteady, but the direction in general is maintained. Patient cannot walk on a line nor backwards. On standing with eyes open he first reels, then constantly falls backwards: on closing the eyes he immediately falls backwards. Patella tendon reflex about normal; the left rather stronger than the right. No foot clonus.

Cutaneous sensibility of the whole body is normal. Accurate examination of the muscular sense, so far as practicable, reveals no derangement. Urine normal in quantity; sp. gr., 1020; acid; contains no abnormal constituent.

From Aug. 27th it was noted that the patient slept well: on Aug. 30th he slept almost throughout the whole day and night.

From Sept. 1st to 6th patient was very restless at night and complained of severe headache; in the daytime he mostly slept.

Sept. 7th.—Headache ceased; no change in the objective indications. On the 13th and 14th patient vomited, during nausea, clear sour fluid in which free lactic acid was found, but no free hydrochloric acid (smaragd-green test).

Sept. 9th.—In the night convulsions with loss of consciousness occurred, causing patient to fall out of bed: he vomited twice at the end of the fit. In the morning he complained of violent pains in the head and eyes. No disorder of consciousness.

Sept. 16th.—No vomiting. Consciousness not impaired; cophosis has increased.

Sept. 18th.—Hearing improved; opthalmoplegia more intense; both the superior recti are functionless, the left abducens also is completely paralysed, the right abducens is paretic. Diplopia absent. The eyelids are raised to only two-thirds of the normal: pupils, of medium size, react very indistinctly with light and during accommodation.

Sept. 24th.—Patient's psychical state has changed; he has become very taciturn, often gives curt answers. The vertigo has increased. On being raised in bed he feels dizzy; moreover, he complains of weakness in both legs. At noon he had an attack of tonic spasm of the lower extremities, with complete loss of consciousness; the pulse was strong, 84 per minute; respiration 18; facial expression, apathetic; eyelids half closed; strabismus seemed to have vanished; pupils, small, reacted with light. After a duration of five minutes the muscular rigidity subsided; patient endeavoured to sit up, but fell backwards. Sensorium is dulled; on loud calling he replies "Hunger" to all questions.

Sept. 25th.—Intellect again completely clear; patient complains of a feeling of continuous severe vertigo; lies quietly in bed. A similar condition during the next few days. No more fits.

Oct. 4th.—Repeated vomiting, with constant vertigo. Distinct paralysis of oral branches of right facial.

Oct. 8th.—A gradually increasing dementia is noted in patient; he complains less of dizziness and headache. Strabismus exists unchanged; pupils are equal, react very sluggishly. The derangement of the ocular muscles shews no increase. Obvious unilateral facial palsy—right. The functions of the remaining cranial nerves undisturbed. The circumference and strength of the upper limbs have diminished; squeezing power of each hand is very feeble; with the right hand patient holds an object very badly and insecurely; a better result is obtained with the left. The lower extremities can be raised in bed and held up for a considerable time; slight passive resistance however cannot be overcome. Gait has become much worse: patient reels to and fro most violently, and falls backwards. Patellar reflex not exaggerated; foot clonus absent. Disorders of general sensibility nowhere present.

Oct. 10th.—Patient again is somnolent; makes no response to loud calling. Now and then he cries for food. Voids the rectal and vesical contents into his bed. To the previously described ocular affections there is added horizontal nystagmus.

Oct. 16th.—Patient can no longer stand alone; he falls to one side and backwards; locomotion is quite impossible.

Oct. 20th.—Intellect is again free. Audition is entirely abolished; one can only communicate with him by signs: visual power also seems to be lessened; patient makes obvious efforts to see clearly. Ophthalmoscope shews consecutive atrophy of the optic nerves. The papillæ are pale and have a porcelain-like appearance; their margin obliterated; arteries narrow; retinæ atrophied; irregular red patches, evidently due to hæmorrhages undergoing absorption, are seen in them.

Oct. 25th.—Pulse frequency has sunk from 64 to 56 per minute. Respirations 14; a deep, often jerky, inspiration is a striking feature of the breathing. Ptosis of left eyelid has become distinct.

Oct. 27th.—Patient has roused up; inquired after his condition, asked how much longer he would have to remain in hospital; seemed also to hear rather better. Examination of the auditory function could not be made either by speech, watch or tuning fork.

Nov. 1st.—Intense dizziness reappeared. Slight arhythmia of pulse.

Nov. 4th.—Ptosis is bilateral and equal; nystagmus, which was absent during last few days, is again present.

Nov. 8th.—Patient sees very imperfectly; he gropes after persons who stand near him.

Nov. 12th.—Declares that he can see nothing; apparently he cannot distinguish between light and darkness. Hearing is again greatly impaired. Pulse arhythmic, small in volume, slight in tension, frequently fluctuates between 84 and 52 during examination.

Nov. 13*th.*—Rigidity of all the extremities, lasting one or two seconds, associated with unconsciousness, occurred. The objective condition was subsequently the same as before. No further change in patient's state until Nov. 30th. Now and then were complaints of headache and dizziness. Vision and hearing almost extinct.

Nov. 30*th.*—Patient lies quite apathetic; his only utterance is a request for food. The ocular movements are in general unchanged, only paralysis of the left superior oblique is superadded. Right pupil somewhat smaller than the left; the right acts very sluggishly, the left not at all.

Dec. 2*nd.*—Patient is somewhat more lively; enquires about his discharge from hospital; often asks for the most various kinds of food.

Dec. 12*th.*—Vomited once after too hasty and free ingestion of food.

Dec. 16*th and* 18*th.*—The same.

Dec. 19*th and* 20*th.*—Lies with his head retracted. No stiffness or pain in the neck. Patient utters much confused, incoherent talk.

Dec. 21*st.*—In the afternoon he was seized with intense dyspnœa. Temperature suddenly rose from 36 to 38.5 and 39. Death ensued during slight clonic spasms of all the limbs.

Sectio Cadaveris.—Skullcap capacious, asymmetric through considerable bulging of the left parietal bone, 53 cm. in circumference, 17.5 cm. in artero-posterior, 15.5 cm. in bi-parietal diameter; very thin, compact. Several deep cavities, varying in size from a millet seed to a hempseed, on internal surface of frontal bone, the smaller involving only the inner table, one of the larger the whole thickness. The sutures are reddened. Dura tensely stretched, its inner surface smooth. The brain greatly swollen; convolutions flattened. Inner membranes very delicate and pale. On the convexity of the frontal lobes several Pacchionian granulations. At the base of the brain the region of the infundibulum projects in semi-globular form, and fluctuates; the region of the sella Turcica deepened by pits. Deep depressions in the anterior and middle fossæ of the cranial base. The pons greatly flattened, also the penduncles, optic tracts and nerves and the chiasma. Lateral ventricles enormously dilated and filled with clear colourless fluid, by which the softened macerated substance of the hemispheres is reduced to the thickness of one or two fingers. The ganglia much flattened. Posterior crura of the fornix firmly adherent to the optic thalami. Foramen of Monro enlarged to the size of a sixpence. The third ventricle greatly expanded, likewise filled with clear serum. The ependyma thin and penetrated by somewhat dilated vessels.

At the situation of the corpora quadrigemina is found a tumour the size of a small apple; it is coarsely lobulated, greyish red, moderately soft, and appears to be richly vascular. The cut surface is granular and shews a somewhat acinous structure with fissural cavities. The tumour bulges downwards into the moderately dilated fourth ventricle. Posteriorly it compresses the cerebellum and its crura. It flattens the aqueduct of Sylvius, thereby completely separating the fourth from the third ventricle, into which latter it projects upwards in the form of a hemisphere. The microscopic investigation of this tumour discloses a papillomatous epithelial growth, probably originating from the choroid plexus.

In the foregoing clinical history are recorded both the symptoms which in my opinion bear the chief importance for the diagnosis of a tumour in the quadrigeminal region, and the combination of which renders the diagnosis possible. *The one of these is the ataxy; or, more correctly, the existence of an unsteady, reeling gait.*

What is the frequency of abnormalities of gait in quadrigeminal tumours? Is this symptom constant; or if absent, can certain grounds for this absence be recognised? Lastly, does it arise from lesion of the corpora quadrigemina themselves, or from implication of contiguous structures?

To the published accounts of these tumours already mentioned must be added those by Bristowe,[1] Ferrier,[2] and Thomas W. Fischer,[3] also three others communicated by me. We have then altogether eighteen cases in which either the corpora quadrigemina alone, or in conjunction with neighbouring parts, were affected by a tumour mass. The circumstance that the corpora quadrigemina were simply compressed by a neighbouring tumour, *e.g.*, of the pineal gland without any morbid growth of their own tissue may at present be left out of consideration.

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[1] BRAIN, vol. vi., p. 167. [2] Ibid, vol. v., p. 123.
[3] Amer. Jour. of Ins., Jan., 1885.

Of twelve of these eighteen patients it is expressly stated that they had a reeling gait, walked unsteadily, hesitated, went like one intoxicated, or could not walk without assistance. The frequency of this phenomenon—the most superficial tabulation shows it to be present in two-thirds of the cases—needs no further emphasising. It becomes even more striking and characteristic if the cases be more closely analysed; especially the six in which it was absent or not mentioned. I have already given a critique of one of the latter. A patient of Henoch's, a phthisical infant, fifteen months old, apparently was totally unable to walk; at any rate no mention is made of the locomotive capacity or the kind of gait. The case of a boy, aged three years, who suffered from tuberculous lung disease, reported by Steffen, is similarly defective. The anatomical description in a case of Rosenthal's is so brief that it is impossible to understand therefrom, whether the corpora quadrigemina themselves were really affected by the tumour—"On the corpus quadrigeminum a medullary neoplasm, the size of a nut, which, reaching to the middle commissure, thrust apart the two optic thalami."

In a case observed by Gowers, destruction was limited to one of the anterior pair of the corpora quadrigemina; we shall revert to this significant case. In Hirtzo "lay a lipoma with two-thirds of its under surface on the right corpus quadrigeminum and geniculatum, the other third pressing on the left corpora quadrigemina"—evidently the substance of the parts in question was not destroyed. Lastly, Pilz's case was an imbecile child, three years of age, suffering from tubercular phthisis, and greatly emaciated; not a word is said in the clinical history concerning its ability to walk during the time it was under observation, probably because the patient was too ill to be out of bed, and thus the condition could not be ascertained. In another case of Henoch's recorded by Bernhardt, but which evidently must be excluded, a large tubercle was found *below* the left corpus quadrigeminum, extending downwards in the substance of the pons.

A strict analysis therefore results in disallowing the conclusion that ataxy was absent in the six or seven cases above cited. These cases must be estimated neither in the positive nor in the negative sense; they are simply useless for affording inferences relative to the symptom in question, either because examination of gait was not made or could not be made, or because the corpora quadrigemina were not directly implicated, or finally, because the description is too inaccurate. The important case of Gowers takes a separate position and also cannot be assigned to the negative side, as will be subsequently shown.

On the other hand all the available cases in which both pairs of the corpora quadrigemina were actually diseased present the symptom of vertigo, of unsteady gait. I think therefore that one is entitled, on the ground of the existing clinical observations and pathological records, to say that the unsteady gait is a constant symptom in disease of the whole quadrigeminal mass.

In Gowers' case nothing is noted of co-ordination disturbances. It is true that the patient complained for some weeks of severe headache; on examination he was in a state of stupor. Death occurred in the course of six or seven weeks and it is not evident whether the "stuporous" patient was generally out of bed. But in any case the *sectio* disclosed only a very partial destruction of the corpora quadrigemina: "The left corpus quadrigeminum anticum was intact, the right anterior destroyed in its inner half; the right posterior was quite normal, the left posterior flattened by the pressure." Strictly taken, one may only conclude from this case that with a solitary lesion of the anterior pair (nates), so long as the hinder pair remain uninjured, disturbances of co-ordination are absent.

Now it is of decisive importance to know whether the derangements of co-ordination are in reality conditioned by the lesion of the corpora quadrigemina themselves. Bernhardt is not of that opinion, for he emphasises the fact that, in the six cases collected by him, the tumour projected backwards into the fourth ventricle or the median region of the cerebellum. One might also object that usually a considerable secondary hydrocephalus (hydrops ventriculorum) has existed, in which affection, as experience teaches, there also may be a reeling gait.

On the other side it may be stated that there undoubtedly are tumours which, rigidly limited to the corpora quadrigemina, in no way involved the cerebellum or fourth ventricle, and yet were associated with co-ordination disturbances. This happened in one of my cases[1] in which at the site of the corpora quadrigemina there was a hard tumour, the size of a hazel nut, pretty sharply circumscribed, which left the cerebellum and fourth ventricle wholly intact. Such cases undoubtedly show that the last mentioned portions of the brain must by no means be imported for the explaining of the functional disturbances. The other objection, viz., that the symptoms originate from the hydrocephalus, seems to me to be untenable, for the following reasons. If the hydrocephalus be the cause of the disturbances of co-ordination, it must have attained a very considerable degree. In that case, other symptoms of increased pressure on the brain must be expected. As a matter of fact such do occur, but—and this is of decisive moment, according to the proof of accurately observed cases—not until advanced periods of the disease; whereas in many tumour cases *defect of co-ordination in walking was the first perceptible symptom.* Thus in the previously detailed case, the first abnormality noticed after the fall was an unsteadiness of gait; headache, vomiting, ocular troubles, &c., were much later in developing. Another of my cases followed a fall on the head in January; in March, vertigo fits and staggering; in the summer, greater impairment of gait, succeeded by other symptoms. This series of symptoms was observed in Koht's patient, but here the cerebellum was additionally involved, consequently the case is not unequivocal, although Kohts, in conformity with Recklinghausen's research, declares that the tumour took its origin from the hinder pair of the quadrigemina bodies. If this latter fact may be considered reliable, great importance must be attached to it, especially when compared with Gowers' case, in which there was destruction only of one anterior body, without ataxy; whereas in Kohts' case destruction of the posterior pair was associated with severe ataxy.

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[1] *Wiener med. Bldtt*, 1888, No. 7.

It is here necessary to briefly examine a fact upon which Bernhardt lays stress for the support of the view that the defective gait in tumours of the corpora quadrigemina should be assigned to involvement of the cerebellum, not to the former. He points out that in the three cases of tumour of the pineal gland, cited by him, no mention is made of the symptom just alluded to, although in each the corpora quadrigemina were more or less affected.

As opposed to this it must be remarked that there are several cases of pineal tumour with concomitant lesion of the corpora quadrigemina in which the symptom existed. In the year 1885 alone three such cases were added to the literature (Feilchenfeld, Pontoppidan, Fisher.) In one of Bernhardt's cases (Blanquinque) it is noted "compression of the corpora quadrigemina," (not alteration of their tissue), "inability of the legs to support the body;" in the second (Nieden), where, it is true, gait troubles were not present, "the parts bordering the third ventricle were somewhat flattened by pressure, but, excepting the superficial portions of the anterior pair of corpora quadrigemina, their structure was uninjured;" this also is susceptible to a criticism presented above. In the third case (Massot) nothing is said of any implication of the quadrigeminal bodies and there was absence of any ataxy.

From analysis and comparative examination of the whole of the cases it results that in substitution of the total corpora quadrigeminal tissue by a tumour, defective co-ordination, an unsteady reeling carriage of the body during locomotion and station, is a constant symptom; and that this symptom depends upon the affection of the corpora quadrigemina themselves, not upon other parts of the brain being involved, nor upon secondary conditions such as hydrocephalus.

Concerning the nature of the co-ordination disturbance, it is displayed as already many times mentioned by an unsteadiness in walking and standing, a stumbling and reeling, altogether comparable to the staggering of a drunken man, or to that which appears in diseases of the cerebellum or its vermiform process. It has no similarity to the ataxy of tabes. The upper extremities are completely free, only the gait and the equilibration of the body while standing, are impaired.

It is needless to say that this ataxy is not pathognomonic of disease of the corpora quadrigemina: it may occur in disease of the vermiform process, of the pons, of the corpus callosum, in hydrocephalus, in some cases of large tumour in the cerebral hemisphere with great augmentation of intracranial pressure, &c. I would attach a diagnostic meaning to it only when it appears as the *first* symptom, for then as a rule the point for decision will be whether the lesion occupies the vermiform process or the corpora quadrigemina.

What guides are there for solving the question in a given case? *This second circumstance* which enables lesion of the corpora quadrigemina, or as one should more cautiously say of the quadrigeminal region to be diagnosed, *is the appearance of paralysis and paresis in the territory of the ocular nerves*, especially of the nervous oculomotorius.

I have already ('Localisation of Diseases of the Brain') expressed myself concerning this point. Reinhold[1] a few years ago published some observations on the ophthalmoplegiæ in tumours of this region. Having quoted those articles for reference I will here merely state the opinion I have gained from the newly added material.

The cases are divided into two groups: (*a*) those in which ophthalmoplegiæ were absent; (*b*) those in which such paralyses were present.

This one fact, that occasionally the ocular muscles are not paralysed and not even paretic, as was notably the case in one of my patients, must be amply sufficing for the conclusion that the lesion of the corpora quadrigemina themselves does not directly, and as such, cause the paralysis of the eye muscles. Rather am I of the opinion that the involvement of the eye musculature is conditioned by a simultaneous lesion—compression or direct invasion by the tumour —of the region of the nuclei of the oculo-motor nerves. I now correct my earlier view, that the disease of the corpora quadrigemina, or especially of the hinder pair, is the immediate cause of the ophthalmoplegia, and I share Reinhold's opinion that the ocular nerve troubles are to be referred to the nuclei and radical fibres of those nerves, not to the ganglia of the corpora quadrigemina.

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[1] *Deutsches Archiv. f. Klin. Med.*, bd. xxxi., S. 1.

Although the existence of ophthalmoplegia alone is of course an inadequate basis for the diagnosis of a tumour of the corpora quadrigemina, it has in my judgment a highly important significance for that localisation, particularly if it be *conjoined with the above described characteristic disturbances of gait.* These associated symptoms would then indicate that the disease which occasioned the abnormal gait must have been so located that it very easily encroached upon the nuclei of the ocular nerves, injuring them directly by destruction of their histological elements, or functionally by compression. Such a localisation is only presented in the quadrigeminal region—in the neighbourhood of the aqueduct of Sylvius.

The special characters of the ophthalmoplegia in these cases are, inequality in the degree of the paralysis, especially in the early periods and in the extent of its distribution. Usually a difference between the two sides can be detected—a certain movement of one globe being merely defective; of the other totally annulled. In the later stages however the paralysis may be equal bilaterally.

Further, it is usual for only some parts of the oculo-motor nuclei to be affected, most commonly those related to the superior and inferior recti; occasionally the lateral movements of the eye are quite abolished, or ptosis may be the first and most marked symptom. Lastly, it may happen that the eye is almost completely motionless, as in primary atrophic nuclear paralysis of the ocular nerves; yet I may remark that in the ophthalmoplegia accompanying tumour of the quadrigeminal bodies I have never observed such entire immobility of the eyes as occurs in the former affection. Theoretically it is possible, but it seems that death takes place before its complete development.

The characteristic in the clinical picture of the ophthalmoplegia connected with these tumours accordingly is the variance in the number of muscles attacked, and in the degree of their palsy.

Sometimes nystagmus, without paralysis in the ocular muscles, has been observed. Perhaps one may not be at fault in assuming that this is a sign of irritation of the nerve nuclei or root-fibres.

Whether an isolated palsy of the N. trochlearis or abduceus (with the defect of gait) can claim a diagnostic meaning is not yet determined.

In apposition to the old view that the corpora quadrigemina are specially related to the visual sense, I have already stated that clinical experience does not support that opinion; Wernicke[1] and Reinhold have expressed themselves to the same effect. Some cases of quadrigeminal tumours were free from disturbances of vision, or, if such were present, there were complications, especially choked disc or optic neuritis with consecutive atrophy, which not only made any decision concerning the relationship between the corpora quadrigemina and the visual faculty impossible, but must themselves be considered as the cause of the amblyopia and amaurosis. Sometimes they may ensue when the beginning of visual troubles coincides with commencing atrophy of the optic nerve, after other symptoms pointing to disease of the corpora quadrigemina have long existed.

The statement that vision or visual acuity may be unimpaired although the corpora quadrigemina be wholly destroyed, can be made without fear of contradiction.

The reaction of the pupils is so various in the individual cases that no definite rule is recognisable.

Disorders of any other kind—motor, sensory, or vasomotor—do not occur, as direct results, in diseases of the corpora quadrigemina; were such observed, there always was implication of other parts of the brain, or hydrocephalus—which so frequently accompanies tumour of the corpora—often with enormous expansion of the lateral ventricles and heightened intra-cranial pressure.

Recently, the relationship of the auditory nerve to the corpora quadrigemina has been referred to, but hitherto the existing clinical material has not admitted of any definite opinion being formed.

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[1] 'Text Book of Diseases of the Brain,' vol. iii.

The substance of the foregoing discussion may be summarised in the following propositions: in a given case in which the signs point to the existence of a cerebral tumour there are grounds for localising it in the corpora quadrigemina, or in the region of the corpora quadrigemina, if the following symptoms be present—(*a*) an unsteady reeling gait, especially if this appear as the first symptom; (*b*) associated with this gait, ophthalmoplegia existing in both eyes, but not quite symmetrically nor implicating all the muscles in equal degree.

CEREBRAL LOCALISATION IN ITS PRACTICAL RELATIONS.

BY DAVID FERRIER, M.D., LL.D., F.R.S.[1]

THOUGH immediate practical utility is no true criterion of the value of any scientific discovery, yet to be useful towards the mitigation of suffering, or the preservation of life, is a consummation which we naturally wish to see achieved by every new addition to our physiological and pathological knowledge. The question before us is, Whether, and to what extent, the doctrine of cerebral localisation is, or is likely to be, of practical avail in the sense above indicated? This question may be conveniently discussed under the three following heads:—

1. Is our knowledge of the functions of the human brain and of the localisation of cerebral disease sufficiently advanced to enable us to determine with a fair measure of accuracy, the locality and nature of disease affecting the cerebral hemispheres?

2. May surgical operations be undertaken on the brain and its coverings with as great safety as any of the major operations in surgery?

3. What diseases and conditions may be considered as justifying or demanding surgical interference, with a view to their removal or amelioration?

I.—In reference to the first head, I think I may say, without fear of contradiction, that within the last twenty years our knowledge of the functions of the brain and the methods of diagnosis of cerebral disease, have made enormous strides. Nor will it be denied that this advance has been consecutive to, if not admittedly due to, physiological experiment. For even if it be said, and with justice, that the principles which guide us in the diagnosis of cerebral disease are those which are based on clinical and pathological evidence in man rather than on the facts of experiments on animals, it is none the less true that, apart from a few empirical generalisations and brilliant hypotheses, the doctrine of cerebral localisation first entered on the stage of demonstration and prediction with the experimental researches begun by Fritsch and Hitziz, in 1870. It is since this time that the facts of clinical medicine have been capable of being read intelligently, and that order has been gradually evolved out of what was previously almost chaos and confusion. There is still, however, considerable diversity of opinion as to the explanation of many clinical facts, and the application of the doctrine of cerebral localisation to the diagnosis of the nature and seat of disease has not always been verified in practice; but it is generally admitted that, even when errors have been committed, it is not the principle itself, but its application, that has been at fault. It is not necessary, before this audience, nor would it be possible for me in the brief compass of a few pages, to examine the evidence, or to discuss in detail the principles of the localisation of cerebral disease. I will therefore only state, very shortly, some of the conclusions which an analysis of the clinical facts appear to me to warrant.

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[1] Read before the Neurological Society, Dec. 20th, 1888.

The region, lesions of which are perhaps the most common, and most easily determined, is the Rolandic zone or motor area—so called because disease situated here invariably leads to motor disorders, spasmodic or paralytic. One of the most significant indications of cortical disease in this region is the occurrence of unilateral spasms—appropriately termed Jacksonian epilepsy—limited to the leg, arm or face; or if not altogether limited, commencing always, or nearly always, in the same part, and invading other muscular groups in a certain definite order. If the spasms begin in the face, they next attack the arm, and then the leg; if they begin in the leg, they attack the arm next and the face last. These attacks are not necessarily accompanied by loss of consciousness, though this not unfrequently happens when the spasms have become general, and pass also to the opposite side. A mere irritative lesion does not necessarily imply demonstrable organic disease, and the starting-point of the irritation may be elsewhere than in the part discharged. But if following these limited spasms, paralysis of motion should occur in the parts formerly convulsed, *i.e.*, if the monospasm give place to a monoplegia, and still more so if a succession of monoplegiæ should result in a general hemiplegia, then we may with certainty diagnose organic disease of the Rolandic zone of the opposite cerebral hemisphere. If the leg is specially affected, the lesion is in the upper third of the Rolandic convolutions; if the arm, in the middle third of the Rolandic convolutions; if the face, in the lower third of the Rolandic zone. And we may more precisely localise the lesion in the upper or lower half of these divisions respectively, according as the proximate or distal movements are more particularly affected. Lesions of the lower facial region in the left hemisphere are almost invariably associated with motor (Broca) aphasia.

According to the extent of the destructive lesion, the paralysis is temporary or permanent—in the latter case followed by descending degeneration in the pyramidal tracts. The electrical reactions of the paralysed parts are not appreciably modified. In the great majority of the recorded cases of cortical paralysis, sensation has been found unimpaired; but, on the other hand, a considerable number of cases have been put on record, in the which, with lesions of various kinds (including tumours) implicating the motor zone, there has been paralysis not only of motion, but also of sensation in a greater or less degree. Very divergent views have been expressed in reference to the interpretation of these facts. I have maintained—and a similar opinion has been expressed by Charcot, Nothnagel, &c.—that there is no necessary connection between cortical lesions of the motor zone and affections of sensibility; and I am further of opinion that the motor and sensory centres are anatomically distinct from each other, though functionally and probably organically connected together. Others (Exner, Luciani, &c.) hold that the sensory and motor centres coincide, and believe that cortical motor lesions affect common sensibility as well as motion. Bastian believes that with lesions of the motor zone there is paralysis of the muscular sense; while Nothnagel is of opinion that paralysis of the muscular sense is related, not to lesions of the cortical motor zone proper, but to those implicating the inferior parietal lobule. It is evident from the discrepancy of views thus enumerated that the facts of disease on which they are based are neither uniform nor altogether simple.

I will not here attempt an analysis of the individual cases adduced in favour of this or that hypothesis, but merely apply certain rules which should guide us in forming a decision on these points. Mere frequency, as the records of cerebral disease amply illustrate, is not sufficient to establish direct causal relationship between the obvious lesion and the symptoms exhibited. Whereas paralysis of motion is invariably caused by truly destructive lesions of the motor zone; anæsthesia is only of occasional occurrence in connection with apparently similar lesions. There is no relation between the extent, degree, or duration of the motor paralysis and the impairment of sensation, for there may be the most absolute paralysis of motion with perfect sensibility in all its forms, cutaneous as well as muscular; and the motor paralysis remains when anæsthesia, if any, has entirely vanished. And, on the other hand, in connection with certain cerebral lesions, there may be absolute anæsthesia with practically unimpaired motor capacity. If there were on record one-tenth of the number of cases of destructive lesion of the so-called motor zone without motor paralysis, as there are of similar lesions without loss of sensation, the whole theory of a special motor zone would have to be abandoned. From this, I think it may be concluded that the sensory and motor centres do not coincide, and that the anæsthesia sometimes observed in connection with lesions implicating also the motor zone is in reality due to direct or indirect implication of sensory tracts or centres. A sensory zone proper is not a mere matter of speculation, but a *vera causa;* for it has been demonstrated beyond all question—in monkeys, at least—by my own and the experiments of Horsley and Schäfer, that the falciform lobe is the cortical centre of common sensibility, inasmuch as destructive lesions of this region produce hemianæsthesia on the opposite side of the body. The position of the sensory tract in the posterior division of the internal capsule is also well known, but we are still in need of information with respect to the position and course of the tracts which connect this with the falciform lobe, and those which associate the latter with the motor zone. Many of the recorded cases of anæsthesia in connection with lesions affecting the motor zone can be shown to have directly implicated also the falciform lobe, or the sensory tracts; and we may legitimately assume, even if we cannot always demonstrate, a similar direct or indirect implication in the case of the others. Those who contend for at most only a slight blunting of the sensibility of the fingers, and not of other parts in connection with lesions of the motor zone, should take into consideration that this may be only a portion, or a remnant of a general hemianæsthesia; for when a general hemianæsthesia is passing off, the fingers are usually the last to recover their pristine sensibility, just as they are the last to recover their delicate movements after a general hemiplegia; and when a limb is motionless, cold, œdematous, or contractured, it may be a less delicate instrument of touch, more from imperfections in the instrument itself, than in the centres of tactile perception.

The question of the relation of cerebral lesions to affections of common sensibility is one of considerable practical importance in reference to regional diagnosis and operative surgery. I should regard a hemiplegia associated with hemianæsthesia either as a sign of lesion of the internal capsule, or if (as judged by the other indications above mentioned) invading the cortical motor zone, as a sign of implication also of the gyrus fornicatus, or its connections with the internal capsule.

Subcortical lesions of the motor zone produce symptoms not readily, if at all, distinguishable from lesions of the cortex itself. They are perhaps less frequently so limited, owing to the close relation and convergence of the various tracts towards the internal capsule, though occasionally they have the differentiated character of monoplegia. Theoretically, on experimental grounds, irritative lesions of the subcortical fibres should produce only tonic and not clonic, or epileptiform, spasms of the related muscular groups, but practically this is not a reliable test, inasmuch as these lesions generally cause also cortical irritation and clonic convulsions of the usual type. More frequently, however, in subcortical than in cortical disease there is an absence of that tenderness on percussion or deep pressure which many years ago ('West Riding Asylum Reports,' Volume II., 1874, BRAIN, Vol. I., 1879) I indicated as a valuable confirmation of the regional diagnosis founded on the symptomatology. I have lately had under my care a case of subcortical tumour of the size of a hen's egg situated at the upper extremity of the Rolandic zone, in which no pain whatever could be elicited by the deepest pressure or percussion, over the region where it was supposed to be, and where the autopsy proved that it actually was. Usually, if not universally, lesions of the cortex, if at all irritative in character, are associated with this localised tenderness to percussion, though no pain may be spontaneously complained of by the patient.

Though the clinical facts of irritative and destructive lesions of the post-frontal or oculo-motor zone are in accordance with the data of experimental physiology, they are not of themselves as yet sufficient to furnish precise regional diagnostic indications. The effects of unilatoral destruction are not permanent, and hence an actual destructive lesion of this region may be entirely latent. The same is true of the marginal gyrus. Lesions may exist in all other portions of the hemisphere without producing obvious symptoms.

Lesions of the prefrontal region cannot with certainty be diagnosed from the symptoms of the lesion as such. The irritable dementia not unfrequently observed in connection with such lesions cannot with certainty be distinguished from the general effects of other cerebral diseases, such as tumour, abscess, and the like. When a regional diagnosis is possible, it is founded mainly on a consideration of the symptoms induced by the not unfrequent implication of the structures in the anterior fossa, together with the effects of extension backwards upon the motor tracts.

Lesions of the occipital region may remain latent, but if the lesion is such as to cause extensive destruction of the medullary fibres, or optic radiations of the occipito-angular region—and this would appear to be of specially frequent occurrence in connection with lesions of the mesial aspect and occipito-temporal convolutions (Nothnagel, Seguin)—we get homonymous hemianopsy towards the opposite side. A similar result may, however, be caused by a lesion of the optic tract, or of the corpora geniculata.

A sudden or apoplectiform onset is in favour of cerebral hemianopsy proper. Cerebral hemianopsy pure and simple is comparatively rare. Very frequently it is associated with a greater or less degree of hemianæsthesia (owing to the implication of the adjacent sensory tracts), slight hemiplegia, or monoplegia, and occasional word-blindness. The visual fields are frequently concentrically contracted, and the dividing line commonly diverges away from the fixation point into the blind side. I have suggested ("Cerebral Amblyopia and Hemiopia," BRAIN, vol. iii.) that a line passing exactly through the fixation point is in favour of tract lesion; but I admit that there are statements on record which seem opposed to this hypothesis. The point is one, however, which I think will well bear further investigation. A distinctive test between cerebral and optic tract hemianopsy, which promises to be of great value, has been proposed by Wilbrand ('Hemianopsie,' 1881, page 89). In cerebral hemianopsy, a pencil of light thrown on the anæsthetic side causes the usual bilateral pupillary reaction; whereas if the lesion is in the tract no such reaction occurs. This test is somewhat difficult to carry out in practice, and special care must be taken to avoid the region of the *macula lutea*.

Word-blindness and allied defects in visual ideation indicate destructive lesion of the angular gyrus of the left hemisphere. Not unfrequently irritative lesions of this region cause subjective ocular spectra, or visual hallucinations.

Word-deafness indicates destructive lesion affecting the superior temporal gyrus of the left hemisphere. Total deafness along with word-deafness may be caused by bilateral lesions of the same region.

Lesions of the other portions of the temporal lobe are generally latent. Lesions of the hippocampal lobule calculated to cause irritation, have given rise to subjective olfactory sensations. Assuming that there were facts indicative of disease of the hemisphere rather than of the olfactory nerves or tracts, such subjective sensations would be in favour of lesion implicating the hippocampal lobule.

Abstracting from traumatic lesions the diagnosis of the *nature* of the disease, whether embolism, thrombosis, hæmorrhage, abscess, syphilis, tubercle, or other cerebral tumours, will depend on a consideration of various factors and symptoms which I do not purpose here to enter upon.

The diagnosis is, however, not always easy, for even the most pathognomonic symptom, such as optic neuritis in cerebral tumour, may occasionally be absent.

In reference to cerebral tumour, in particular, the attention of neurologists is greatly needed towards the discovery and formulation of signs and symptoms which will serve as better guides than we at present possess, in determining the exact nature of the tumour, and whether it is an isolable or infiltrating growth. The diagnosis is at present unfortunately in many cases only possible after death, or during the operation undertaken to remove it.

II.—*May surgical operations be undertaken on the brain and its coverings with as great safety as any of the major operations in surgery?*

In reference to the second head, the opinions of surgeons have been very much divided. While some, up to a comparatively recent date, have looked upon trephining as a most dangerous and unjustifiable operation, others have regarded the operation *per se* as attended by comparatively little risk; attributing the fatal results (unfortunately all too frequent) to the conditions under which the operation was undertaken. Walsham (St. Bartholomew Reports, 1882-3) has analysed the results of 686 cases of trephining, both for cranio-cerebral injuries and traumatic epilepsy, and arrives at the conclusion that though the published statistics would appear to show that trephining *per se* is a very dangerous operation, being followed by a mortality of 50 per cent., yet this is not a legitimate deduction from the facts. In very few of the 269 deaths occurring in these 686 cases, could the trephining be truly regarded as the real cause of death—this being the condition which the operation was undertaken to cure. In 122 cases, where the disease was not of such a nature as to endanger life at the time, the mortality was only 13, or 10·6 per cent. This mortality, however, he is of opinion might be further reduced, provided proper antiseptic precautions were adopted and the membranes not wounded. Amidon ('Annals of Surgery,' vol. i., 1885), from a study of the facts of 115 cases, comes to the conclusion that, apart from symptoms endangering life at the time of the operation, a mortality of only 3·2 per cent. could be properly attributed to the operation itself. Bluhm (*Archiv. fur Klin Chir.*, 1876), analysing 331 cases of trephining before the era of antiseptics, fixes the mortality from all causes at 44 per cent., while Seydel (*Antiseptik u. Trepanation*, 1886) says that under antiseptic treatment the mortality in 289 cases, from all causes, was only 15·5 per cent.; while the mortality from the simple operation of trephining itself did not amount to more than 1·6 per cent. Even those surgeons who, like Walsham, have regarded trephining as a comparatively safe operation *per se*, have made the proviso that the membranes should not be opened, or the brain itself operated upon. No one, so far as I know, had, up to a very recent date, advocated the deliberate opening of the cerebral membranes and operation on the brain itself, for the relief of diseases localised by their symptomatology, entirely irrespective of traumatic influence. I was led to advocate this by a comparison of the results of the experiments which I had made on the brains of monkeys without antiseptics, with those obtained by Prof. D. F. Yeo and myself, under strictly antiseptic precautions, as published in the *British Medical Journal*, 1880. Whereas in the first series of experiments encephalitis, or meningocephalitis was the invariable, and almost always fatal result, in the second series no such result occurred (except once, when the antiseptic treatment was undoubtedly interfered with); and even after the most formidable, and occasionally twice or thrice repeated removal of portions of the brain, the animals continued in perfect health and free from first to last, from fever or other constitutional disturbance. It could not be said that experiments on monkeys were not comparable to those on man, or that these animals could bear operations without the risks attendant on similar operations in human beings, for the first series of experiments showed conclusively that monkeys are liable to precisely the same dangers as those which are the chief cause of death in man. I, therefore, having frequently before suggested, ventured formally (*Medical Chirurgical Transactions*, Oct., 1883) to advocate operative procedure in such diseases as cerebral tumour, in respect to which all therapeutic remedies had hitherto proved unavailing. Though MacEwen seems to have arrived at the same conclusion, and had actually ("Address in Surgery at the Meeting of the British Medical Association in Glasgow," *British Medical Journal*, Sept., 1888) operated successfully, not only in several cases of traumatic effusion of the blood, but also in two cases of cerebral tumour—one of which was the recurrence of a previously excised tumour of the left orbit, and the other a syphilitic nodule in the paracentral lobule—the first published case of removal of a cerebral tumour, indicated and localised purely from the symptomatology, altogether apart from external indications, was Dr. Bennett's case, operated upon by Mr. Godlee (*Lancet*, Dec. 20th, 1884). This case I had the opportunity of seeing, with Dr. Bennett, and of confirming his diagnosis and supporting him in the treatment which he had resolved on. Though the case terminated fatally, this was due to secondary, and obviously avoidable, inflammation, and so far it demonstrated that the mere removal of a cerebral tumor was not in itself a necessarily dangerous or fatal proceeding. This case was the chief means of stimulating the attention of the profession to the surgical treatment of cerebral diseases. Since then many similar operations have been performed, and a large measure of success has been achieved. MacEwen reports successful results in eighteen out of twenty-one cases operated upon by him. Of the nine cases of which he gives details, two were of cerebral abscess—one of which proved fatal; four were tumours, including one cyst; one was a traumatic lesion of the angular gyrus entirely localized by symptomatology, (viz., word blindness and homicidal impulse); and two were hæmorrhagic extravasation.

Horsley (*British Medical Journal,* Oct. 9th, 1886 and April 23rd, 1887) has, at this hospital (Queen Square), operated successfully three times for cerebral tumour, though in one of the three, recurrence took place, followed by death six months afterwards. He has five times excised irritative lesions of the cortex, originally due to traumatic influence; and once successfully evacuated a cerebral abscess (*British Medical Journal,* March 10th, 1888). Keen (*American Journal of the Medical Sciences,* Oct. and Nov. 1888) has successfully removed a large cerebral tumour, weighing over three ounces; and has twice successfully excised irritative lesions of the motor cortex. Durante (*Lancet,* Oct. 1st, 1887) has recorded a case of successful removal of a tumour from the left anterior fossa. Weir has successfully removed a tumour from the motor cortex in a case diagnosed by Seguin (*American Journal of Medical Sciences,* July and Sept., 1888). Markoe (*Medical News,* Nov. 5th, 1887) successfully removed a cyst, or tumour, probably due to injuries received eight years previously; and Kendall Franks has recorded a similar case operated upon four years after the receipt of the injury (*British Medical Journal,* April 9th, 1887). A case of successful excision of a portion of the motor cortex for Jacksonian epilepsy has been recorded by Lloyd and Deaver (*American Journal of Medical Sciences,* Nov., 1888); another by v. Bergman (*Archiv. fur Klin. Chir.,* Vol. 36., 1887, p. 860); while Hughes Bennett (*British Medical Journal,* Jan. 1st, 1887) reports a case of opening the dura mater, and probing of the cortex (in the region of the angular gyrus) the supposed seat of the irritation in a case of epilepsy with visual hallucinations, supposed due to a blow received six years previously. Edmund Owen (*British Medical Journal,* Oct. 13th, 1888) reports a case of successful removal of a hæmorrhagic extravasation localised by symptoms, apart from external indication; and a case somewhat similar, though guided largely by external indications, has been recorded by Oliver (*British Medical Journal*, Feb. 4th, 1888). In addition to the two cases of cerebral abscess successfully evacuated by MacEwen and Horsley, two others have been recorded by Barker (*British Medical Journal*, Dec. 11th, 1886 and April 14th, 1888), and a fifth by Greenfield (*British Medical Journal*, Feb. 12th, 1887). In all these five cases the abscess was localised by symptomatology, entirely apart from external indication beyond the fact of the existence of otitis media.

Though I have purposely excluded from this review operations for traumatic abscess, two others might be mentioned in this connection reported by Truckenbrodt and Schondorf respectively (referred to in my paper, *British Medical Journal*, March 10th, 1888). Besides these cases of successful trephining and operation on the brain, two have been reported in which the disease was either not found, or not removable. One of these, a tumour in the cerebellum, has been reported by Weir (*American Journal of Medical Sciences*, Sept. 1888). Death occured two and a half months afterwards, obviously irrespective of the operation. The second was an operation by Heath (*Lancet*, April 7th, 1888) for a tumour situated in the anterior fossa, which could not be removed on account of adhesions. The patient was alive thirteen months after the operation.

This gives us a list of forty-six cases in which the skull has been trephined, the dura mater opened, and the brain itself operated upon for tumours, abscesses, irritative lesions, and more or less distant results of traumatic injury in which the indications for the trephining have been mainly given by the symptomatology in accordance with the principles of cerebral localisation, and all successful quâ the operation itself. But against this record must be placed a list of unsuccessful cases in which death has resulted either within a few hours from shock or a similar condition, or within a fortnight after the operation from septic inflammation, or from other causes not perhaps strictly attributable to the operation itself.

Three fatal cases of removal of tumour from the cerebellum have been reported respectively by Bennet E. May (*Lancet*, April 16th, 1887), Suckling (*Ibid*, October 1st, 1887), and Horsley (*British Medical Journal*, April 23rd, 1887). Of unsuccessful removal, or attempts at removal, of cerebral tumour, one is reported by Hirschfelder (*Pacific Medical and Surgical Journal* April, 1886), death in this case being due to septic inflammation; a second by Birdsall (*Medical News*, April 16th, 1887) death due to shock and hæmorrhage; a third by Hammond (*Medical News*, April 23rd, 1887), death resulting twenty-four hours afterwards; and a fourth (tumour of the skull and brain) by v. Bergmann (*Archiv. fur Klin. Chir.*, 1887, vol. xxxvi., page 829). In this case death occurred on the fifth day, from apparently, cerebral œdema. To these I have to add two cases of exploratory trephining for cerebral tumour under my own care. In the one of these the operation, performed by Sir Joseph Lister, was for tumour in the right anterior fossa, which, however, was too deeply situated to be reached. The patient was in a state of coma at the time of the operation, and death occurred on the eighth day under gradually increasing coma, but without inflammation. A second case, operated upon by Mr. Rose, was a tumour of the right temporo-sphenoidal lobe. The patient was *in extremis* when operated upon, and the tumour was too deeply situated to be removed. In this case death occurred within twenty-four hours afterwards.

If we include the fatal cases of operation on the cerebellum, which perhaps, however, should be better considered apart, the mortality of the various operations above recorded would appear to be 21·7 per cent.; excluding cerebellum cases, the mortality would be 15·2 per cent. It is possible that other cases, yet to be recorded, may modify these statistics to some extent, but I think we may safely say that the mortality from all operations, including the removal of even large cerebral tumours, will be found to be considerably less than 30 per cent. This mortality will be found to contrast not unfavourably with that which results from some of the major and generally recognised legitimate operations in surgery. I extract the following from the latest edition of "Erichsen's Surgery:"—

The mortality after amputation of the hip joint varies from 70 to 42 per cent.

Amputation of the shoulder joint 49½ per cent.

Ligature of the common carotid 68 per cent.

Laparotomy for intestinal obstruction 80 per cent.

Strangulated hernia (after two days) 40 per cent.

III.—What diseases and conditions may be considered as justifying or demanding surgical interference with the view to their removal or amelioration?

There can, I think, be little question as to the advisability of trephining in primary cranial injuries, with symptoms of compression or localized paralysis or convulsions, with a view to removing depressed fractures, splinters of bone, or hæmorrhagic extravasations, on which these symptoms depend. For when we consider the successful achievements recently reported by MacEwen (*supra cit.*) and Owen (*supra cit.*) and the fact that operations of this kind under antiseptic precautions do not amount to more than 8.6 per cent. (Seydel), and when we remember also that, even if without operative procedure some cases of compression apparently get well spontaneously, yet subsequently, often many years afterwards, the patient may become epileptic or insane, we have good ground for regarding the operation as not only justified, but imperatively demanded as a preventive measure. Nor can there be any question as to the advisability of trephining with a view to the evacuation of traumatic abscess. For though the mortality, even under antiseptics, appears to be very high—amounting, according to Seydel, to 63·6 per cent—yet, as a spontaneous cure is practically unknown, death would be the inevitable result in all cases. Nor will there, I imagine, be any question as to the advisability of operation with a view to the evacuation of a collection of pus from any cause not traumatic, provided that the seat of the abscess can be accurately determined.

The great majority of cerebral abscesses arise in connection with disease of the middle ear; but neither the fact nor the seat of the abscess is at all times clearly revealed by the symptomatology. We can, however, point to some brilliant examples of successful localisation and evacuation of cerebral abscess, within recent times, besides the two successful cases reported by Schondorf (*Monatgsch. für Ohren heilk*, No. 2, 1885) and Truckenbrod (*Archives of Otology*, June—September, 1886), in which the localisation was largely determined from external indications, five others, diagnosed from the symptomatology alone, and successfully treated, have been recorded respectively by Gowers and Barker (*British Medical Journal*, Dec. 11th, 1886); Greenfield (*Ibid*, Feb. 12th, 1887); MacEwen (*Lancet*, March 26th, 1887); Ferrier and Horsley (*British Medical Journal*, March 10th, 1888); and Barker (*Ibid*, April 14th, 1888). There seems good reason for believing that equally successful cases will become more numerous in the future. While, however, there is little room for doubt as to the expediency of primary trephining for cranial injuries and their more or less immediate consequences, the question is different when we come to consider the question of secondary trephining with a view more especially to cure epilepsy or similar affections due to, or supposed to be due to, a cranial injury inflicted at a more or less distant date.

Though trephining for this purpose dates even from pre-historic times, it is by no means settled how far as a curative measure in the true sense of the word, it has proved successful. Apart from the risks of the operation itself—which, according to Billings, has been attended by a mortality of 28 per cent. before antiseptics; but since the introduction of antiseptic treatment estimated by Seydel at 0 per cent.—the proportion of cures of traumatic epilepsy has been estimated by Eccheverria at 65 per cent.; by Walsham at 58 per cent.; and by König at 59 per cent.; and by Seydel at 69·2. In 82 cases carefully analysed by Walsham the primary nature of the lesion, where one for certainty was known to have occurred, was in more than half the cases a fracture, generally compound, with a depression. In the remainder, the injury when known was various—a scalp wound with possible bruising of bone, a contusion of the scalp, or a simple fracture—whilst in many no history was obtained of the primary injury, further than that the patient had a fall, or received a blow on the head many years ago, often in childhood. In all the cases, with but few exceptions, there were some local indications for the use of the trephine. In a large majority there was a depression, or cicatrix, tender or painful, either on pressure or otherwise; whilst in others there was a tender or painful spot without depression or other mark of former injury. In two-thirds of the cases a portion of the bone was found either depressed, or variously altered or diseased. The dura mater in the greater number of cases appeared healthy, but in some was thickened, congested, vascular, adherent or otherwise altered. In sixteen, nothing was found by the operation to account for the epilepsy. Six of these died, and in two, even at the post-mortem examination, no cause for the epilepsy could be discovered. The remaining ten recovered from the operation, and all, with the exception of three, were cured of the epilepsy and other symptoms for which they were trephined. In the three exceptions, two were improved, one was not improved. There is good reason for believing, however, that the number of real cures of traumatic epilepsy is not so great as these statistics would lead us to believe. Cases are too often set down as cured when in reality the patient has only survived the operation, and remained free from fits for the comparatively short period intervening between the operation and his discharge from the hospital. But this may easily be the case after trephining, as after almost any surgical operation whatever; and yet the fits may recur in all their original frequency and intensity after a longer or shorter interval. It would not be safe to count on a cessation of the fits until at least a whole year should have elapsed since the date of the operation without any recurrence. Tested by this standard, there are exceedingly few cases on record in which it can be stated that the fits remained in abeyance after the operation. Of Walsham's 82 cases, I can only find 12 of which this can be predicated. Hence the cures of traumatic epilepsy by simple trephining, without opening the dura mater would be placed not at 58 per cent., but at the much lower figure of 14·6. In three cases which have come under my own observation, in which epilepsy occurred after, and apparently in consequence of injury to the head, and in which there were distinct signs of depression or local tenderness over the seat of injury, trephining was not of the slightest benefit. One of these cases was the boy O. G. H. (referred to by Mr. Horsley, Case 5 (*British Medical Journal,* April 23rd, 1887), who first came under my care in September, 1881. He was then seven years old, and some years previously had received a severe blow on the right side of the head, of which he retained a well-marked scar above, and in front, of the right ear, at a point just anterior to the position of the facial centre. When I first saw him he had just commenced to have fits, beginning, and often confined to the left angle of the mouth. He had one of these in my presence, of the typical Jacksonian type, without loss of consciousness. As the attacks did not yield to medicinal remedies, but on the contrary, tended to spread and become general, he was, at my request, trephined by Sir Joseph Lister, over the seat of injury. No appreciable abnormality could be detected in the bone, or the dura mater, which was not opened. The boy made a speedy recovery from the operation, but the fits were not in the slightest degree affected after the first day. A year afterwards I placed the boy under the care of Mr. Horsley, with a view to excision of the facial centre—the result of which I will relate subsequently. Besides these cases which have come under my own observation, I might easily quote other similar unsuccessful cases, in the recent practice of others. Three such are related by v. Bergmann ('Archiv. fur Klin. Chir.,' vol. xxxvi., 1887), and as the general result of my investigations and experience in reference to the question of trephining for traumatic epilepsy, I would say that unless (besides the mere history of a blow on the head) there is clear evidence of local injury in the shape of a distinct cicatrix or depression, and in addition some signs of localised irritation of the cortex at or near the site of injury, trephining is not indicated, and even then the result is extremely doubtful. The prospects of benefit are much greater when, in addition to trephining the skull, the whole of the cicatrical tissue and irritable portion of the brain cortex are completely excised.

One of the most successful examples of this kind is the case of J. B. (Mr. Horsley's first case *supra cit.*) who first came under my care in 1884. The patient, a lad of twenty, had received when seven years old a compound fracture of the skull and injury of the brain, in the region of the upper part of the left ascending frontal convolution, which had caused a permanent slight degree of right hemiplegia. For five years he had been subject to fits, mainly on the right side, beginning generally with rotation of the head to the right. He was admitted as an in-patient under the care of Dr. Jackson and myself in December, 1884. Between this time and May, 1885, he had an enormous number of fits, not materially influenced by treatment, but which had spontaneously entirely ceased for two months before he was discharged. He came again under my observation seven months later, and as there seemed to be a very distinct relation between the epileptiform tendency and tenderness of the scar, I came to the conclusion, having previously tried counter-irritation over this region without benefit, that it would be advisable to have the whole of the cicatrical tissue excised. This was undertaken by Mr. Horsley who thoroughly removed the cicatricial tissue up to the healthy brain substance, with the result, which he has described, of a complete cure of the epileptic fits and only a slight increase in the hemiplegia. I saw the patient on the 9th of December of this year; he is in perfect health, and has not had the slightest sign of a fit since the date of the operation—a period of upwards of two years and six months.

The successful result in this case would seem to warrant a confident hope that the complete removal of the focal irritation would lead to a complete cessation of the fits in all similar cases. The suggestion, first made by Hughlings Jackson, that in cases of focal epilepsy, whether dependent on organic disease or not, the discharging lesion should be excised, has met with widespread approval, and has led to a considerable number of operations with this object. Many of these have been so recently carried out, that it is perhaps too soon to speak with confidence as to the ultimate issue. But though the results have been on the whole very encouraging, I fear it must be admitted that in some at least, the expectations of permanent benefit have not been altogether realized. One of the most striking cases of that kind is the boy O. G. H. above alluded to. Mere trephining over the seat of the injury having proved unavailing, the right facial centre, the primary focus of discharge, was excised by Mr. Horsley a year after the first operation. Notwithstanding this the fits have not ceased, and having examined the boy the other day (Dec. 3rd), I find that he continues to have epileptiform attacks every night varying in number from three to four up to twenty, and the remarkable circumstance is, that the fits begin as before in the left angle of the mouth.

In two if not three of the cases operated upon by Mr. Horsley (*supra cit.*) the fits have not entirely ceased, even though perhaps modified. A similar unsuccessful case (excision of the Hand centre) has been described by Bergmann (*supra cit.*); and another (also excision of the Hand centre) by Keen (*supra cit.*). In Dr. Bennett's case (probing of the angular gyrus) the fits returned in ten months, but again ceased after excision of the cicatrix of the first wound. It is uncertain whether they have ceased entirely, as the man has been lost sight of; but the probability is that they have not returned, otherwise he would have reported himself. Out of twelve cases, however, on record, the fits have not entirely ceased in six, and sufficient time has not as yet elapsed to judge with certainty as to the ultimate fate of the others. It is of the utmost importance that the causes of failure should be discovered, and if possible obviated. The recurrence of the fits may be due (1) to incomplete excision of the focal irritation; (2) to the neighbouring centres having become irritable and unstable like the original focus; (3) to a habit of discharge being established in the other hemisphere, or possibly in the lower centres. If the cause were the last mentioned it would follow that the operation, to be successful would have to be performed before what we might call an epileptiform habit had become established. If the cause were the second mentioned, the operation of excision would appear to offer little prospect of success, except at the expense of a considerable degree of paralysis. The question will then arise, whether if the epileptiform fits can only be cured by the establishment of extensive hemiplegia, aphasia, or other great impairment of faculty, the operation may not be considered as a greater evil than that for the cure of which it is intended.

I am inclined to think that perhaps all the factors which I have mentioned may occasionally be operative: but it would seem that if the fits recurred in the same muscular groups as before, the chief reason must be imperfect excision of the focus of irritation. This would afford the most reasonable explanation of the recurrence of the fits in the left angle of the mouth in O. G. H., previously alluded to; and a similar explanation is applicable to some at least of the others on record. The lesson to be drawn from this, therefore, would be that in all cases there should be as complete excision as possible of the whole centre from which the discharge proceeds.

As an illustration of the establishment of an epileptiform habit, I would here mention the facts of a case which has been recently under my care at King's College Hospital. The patient, a young woman, aged twenty-five, had received a compound fracture of the skull and injury of the brain in the right parietal region, sixteen years previously, causing permanent hemiplegia of the left side. A year after the injury she began to have left-sided fits, which continued up to last year, when she was admitted into King's College Hospital. In April, 1888, Mr. Rose removed the thickened and depressed edges of the bone and broke up the adhesions, but no portion of the cortex was excised as the parts had undergone such atrophy that there was considerable risk of opening into the lateral ventricle. The fits recurred, and now occasionally affected both sides. As certain portions of the scar still continued tender to pressure, a second operation was undertaken in July, and further portions of bone removed. The fits ceased for five months, but again recurred, this time affecting only the right side; the left side to which previously the fits were confined remaining quiescent.

This case would seem to show that from a long continued irritation limited to one hemisphere, the other hemisphere (or perhaps lower centres) may take on what may be termed an epileptiform habit.

As regards the treatment of cerebral tumours these, in the great majority of instances, lead to death sooner or later, so that the condition must in all cases be considered desperate. Hence the question is not between the relative advantages of this or that mode of treatment, but between the possibility of removal by operation, and certain and, too often painful death. Even if we accept as accurate the statistics of Hale White ('Guy's Hospital Reports' vol. iii., 1886) that, at most, only 10 per cent. of all cerebral tumours are amenable to operation, this is a fact which we may deplore, but it ought not to influence us against the endeavour to cure, if possible, the cases in which the tumour is so situated, and of such a character, as to admit of removal. And such cases are sufficiently numerous to come at some time or other, under the cognizance of almost every physician. Already in a comparatively short space of time, eighteen cases at least have been operated upon; of these, seven, including one cyst, have been successfully removed; of the remaining eleven cases (including three cases of tumour of the cerebellum, five of unremovable tumours—two of them operated upon *in extremis*), nine have died from various causes, including septic inflammation, cerebral œdema, or shock. This gives us a mortality of 50 per cent.—or as it should rather be put, a salvation of at least half the cases.

Contrasted with the older statistics of trephining for all causes, or any of the major operations in surgery, I think we have every reason to regard the surgical treatment of cerebral tumours as having achieved an encouraging measure of success. And there is reason to believe that greater successes may be attained in future, as the conditions of successful operation and after-treatment become better known. No cases at least should die of septic inflammation, to which we owe two of the fatal cases on record.

Operation is advisable as soon as the nature of the disease has been clearly determined, and before the tumour has acquired such dimensions as to seriously impair the vital resistance, and increase the dangers from shock, hæmorrhage or cerebral œdema. Even very large tumours—from three to four ounces in weight—and a corresponding number of cubic inches measurement—have been successfully removed, and that too under conditions theoretically most unfavourable, such as the existence of a state of coma, or semi-coma. Yet the risks are no doubt thereby greatly increased.

Another reason for early operation is the uncertainty as to the nature of the tumour, whether isolable or infiltrating: for while an infiltrating tumour is not absolutely unremovable, yet the prospect of permanently successful removal is greatly diminished if the tumour has already attained a considerable size.

In the case of W. T. (Horsley's case 9, *supra cit.*), an infiltrating sarcoma, several ounces in weight, was successfully removed, and there have been no signs of recurrence up to a comparatively recent date—a period of two years.*

In the absence of definite indications as to the character of the tumour, and as to whether it is situated on, in, or beneath the cortex, trephining is, in my opinion, justifiable as a diagnostic measure; for if the tumour should after all not be removeable, the risks of the operation itself are out of all proportion much less than the evil of allowing a case to perish, which the autopsy might prove to be one which could have been dealt with successfully.

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* I regret, however, to say, that in response to enquiries I made the other day, I have received a letter from Mr. Birch, of Newbury, dated Dec. 17th, which makes me fear that there are some indications of recurring mischief. I have not as yet seen the patient myself, but Mr. Birch writes as follows:—"I had not seen or heard anything of him for some months, until Saturday, the 1st instant. I was then asked to see him, and found that for a few weeks he had been suffering from pain in the head, on the site of the wound. The pain was intermittent, but very severe, lasting about a quarter of an hour, and occurring four or five times a day. I gave him bromide of potassium, and the next morning he said he was better; but while I was sitting by his bed, his eyes turned to the right for about a quarter of a minute and then began to oscillate laterally, rapidly at first, gradually getting slower. The whole attack did not last more than a minute, and while it lasted he could not see. On Monday, the 3rd, I had him removed to the District Hospital, that he might be under better observation. On the afternoon of that day he had another attack in my presence, similar in character, but more severe. He had about two of these attacks daily at the beginning of the week."

Addendum Note, July, 1889.—There is reason to believe that the above attack was due to intemperance. The patient speedily recovered, and when I saw him on May 4th, he was quite well and free from all pain in the head. A week after (May 11th), he accidentally fell down stairs, and sustained fracture of the skull with meningeal hæmorrhage, of which he died on the 16th. The condition of the brain will be the subject of a further note.

[This paper, written more than six months ago, is now, owing to delay in publication, somewhat out of date, as several important cases recently published are not referred to. I prefer, however, for various reasons, to leave the paper in its original form.]

ACROMEGALY.

BY PIERRE MARIE, M.D.,

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Translated from the French original MS. by William Dudley, M.B.

THE disease, the clinical description of which forms the subject of this article, is by no means unknown in England, where it has already been the subject of reports and discussions before the Clinical and the Pathological Societies of London. Among the names of physicians and surgeons who have studied it, we find those of Wilks, Hadden, Godlee, and other able observers. I hope, however, that for the majority of the members of the profession, a formal description may yet prove acceptable, and I have to thank the editor of 'BRAIN' very sincerely, for having kindly furnished me with the means and opportunity of coming forward with such a contribution to medical literature under such particularly favourable conditions.

The first author, to my knowledge, who seems to have observed and described a case of this affection is the surgeon Sancerotti, in 1772; since then, a certain number of observations of the same kind have been published in different countries by various authors, and under the most diverse names—exophthalmic goitre, myxœdema, hypertrophy of the tongue, &c., &c., . . . and "gigantism;" this last designation having been the one most commonly employed, in consequence of the confusion made between this anomaly of development and acromegaly. In 1885 when chief assistant to Professor Charcot, I was able to observe in his ward two cases of this kind, presenting in a high degree the characteristic symptom to be described farther on. Thus I was led to make a special study of this affection, and to endeavour to describe it, bringing into prominence the typical phenomena which characterise it. I became convinced that I had before me a distinct *morbid entity*. I did my best to define it in its sharp outlines. In order to complete my task, I selected one of the most prominent symptoms, viz., *a striking non-congenital hypertrophy of the extremities* (hands, feet, cephalic extremity); I proposed for it the name of *acromegaly* (from ακρον, extremity, and μεγας, large.)

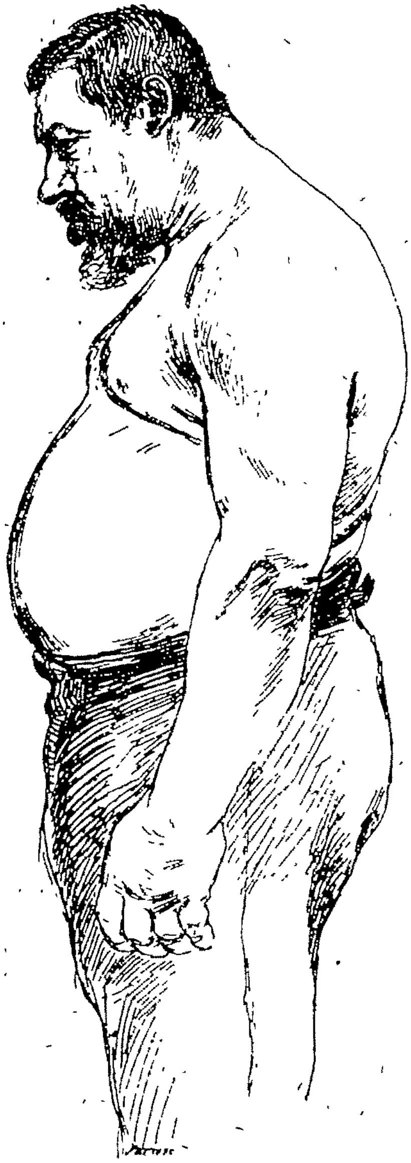
Since this period a certain number of new confirmatory observations have been published and some others which had escaped my first researches have been brought forward; for my own part I have had the opportunity of making an autopsy on one of my first patients, and of studying two new cases; it is one of these last cases, hitherto unreported, that I am now publishing in 'BRAIN.'

CASE.—Baud., age forty-nine, employed in a printing-house. No member of his family (father, mother, brother) has presented any enlargements of the limbs. His father, who is yet living (age seventy-three), is in very good health. The patient himself was very puny at birth (weighed one-and-a-half kilogrammes). At the age of fifteen he had severe typhoid fever. Served his term in the army; has never had any venereal affection (is quite certain on this point). In 1881 had in the scalp over the occiput a very confluent, pustular, very painful eruption; the acute and inflammatory period was of four or five days' duration, he could not sleep it pained him so much, then the pimples dried up.

The patient cannot say exactly since when the limbs have been increasing in size (since he had not attached any importance to it, and had hardly noticed it). This, however, is what he remembers: being a soldier, up to the age of twenty-seven he never wore large shoes, "he was even fitted with shoes rather small than large for his height." Till 1878 no one had ever remarked on the size of his hands; it was only at this time that that began. It seems therefore certain to him that the first symptoms made their appearance between the year 1867 (the date when the patient was discharged from the army) and the year 1878.

At thirty years of age the weight of the patient had already increased a little; he was 80 kilogrammes instead of 64 at 27. But it is especially from 1878 that his weight and size have more manifestly increased; in December, 1887, his weight was 113 kilogrammes; in December, 1888, 106 kilogrammes.

His height (measured barefoot) when the patient was in the regiment, was 1670 mm. At the present time, measured in shoes, (about 2 to 3 cm. in depth of heel), it is 1690 mm.



Present Condition, Jan., 1889.—The appearance of the patient is qute charcteristic (the diagnosis was made in the street); the hands and feet are enormous; the hands present the character stümpy (camard), as well as the fingers; the hypertrophy of the soft parts is such that the patient cannot close his hand in the form of a fist (flexion of the first, second, and third phalanges); all he can do is to flex the first and second phalanges. The tips of the fingers cannot therefore be applied to the middle part of the palm of the hand, but only to the upper part of the latter. The left index finger has had a whitlow (from a splinter of wood), in February, 1888; since then the patient has often experienced at this level the feeling of deadness of the finger. The wrist is thick, but relatively smaller than the hand; the forearm and the arm are equally bulky, without however their dimensions giving rise—as do those of the hand—to the idea of monstrosity; the arm and the forearm are moreover as regards size, in perfect relationship one to the other;

the muscles show very notable development without any hypertrophy; the strength of the patient is great, as a soldier he was the strongest man of his company; since then he has not noticed that he has become weaker out of proportion to his advancing age.

The face is equally characteristic; the nose is large and somewhat pug-nosed. The lower lip is enormous, everted and looks like a hanging pad; the upper lip is rather thick, but is not in this respect comparable to the lower. The lower jaw is very much hypertrophied, the chin forms a marked projection; prognathism is such that the incisors below reach beyond those above from 7 to 8 mm., they are, moreover, neither large nor projecting.

The tongue is very large, very long, rather thick. Articulation of words has become, not difficult, but somewhat confused, probably in consequence of hypertrophy of the tongue and of the soft parts of the mouth.

Neither the eyelids nor the ears are of abnormal dimensions; hair well preserved, rather thick, but has always been so, he says; the beard is rather abundant, slightly curly. The neck is thick and short; there is well-pronounced cervico-dorsal kyphosis, and consequently marked inclination of the head forwards. The larynx is rather large, but it cannot be certainly said to be hypertrophied; the thyroid body cannot be felt sufficiently distinctly for it to be said whether it is normal or not. The voice is strong, the tone of it is not abnormally deep; it is besides very discordant, and it is impossible to make the patient sing the scale so as to measure the limits of it.

The thorax is bulky; the sternum very oblique; the xiphoid appendix very prominent and hypertrophied, makes a very appreciable projection under the skin. The lateral walls of the thorax are a little flattened; the lower part of the thorax moves prominently forward during inspiration. The retro-sternal dulness of Erb has not been able to be determined. The heart is perhaps a little hypertrophied but otherwise seems normal. The pulse is rather small and compressible.

The genital organs present nothing unusual. The penis is rather large, but all the others are of ordinary dimensions. The sexual appetite has never been great and has become lessened during the last few years (it must not be forgotten that the patient is a diabetic). The skin, while not being of a very clear tint, has not however that brown olive colour presented by some patients. Nowhere are vergetures observed, but there is a single growth of molluscum fibrosum on the right shoulder.

Cutaneous sensibility presents nothing to remark; the knee reflexes are present.

Nothing particular as regards the special senses; sight however may have become a little less acute for small print. No ophthalmoscopic examination.

There is very evident dilatation of the veins of the leg, especially in the neighbourhood of the internal malleolus, but only slight varicosity.

The mental faculties are good; the patient has intelligence above his station in life, and he has been self-taught.

He has never suffered from headache—of this fact he is perfectly sure.

The patient's appetite has been excessive, especially for two or three years; likewise his thirst (5 litres on the average, of which 2 litres of wine); to quench his thirst he has to drink much at a time.

Examination of the urine has revealed a very notable amount of sugar. We have before us, therefore, a diabetic, and henceforth it becomes impossible to say if the polyphagia, polydipsia, and polyuria have any relation at all with the acromegaly, or if they do not depend entirely on the diabetes.

Additional Note, May 15th, 1889.—Treatment was instituted in January to combat the diabetes (alkalies, arsenic, diet); the sugar diminished pretty rapidly; now, no trace of it can be shewn with potash-copper solution. At the same time, thirst has diminished, the patient is now much more active, he no longer experiences either lassitude or a tendency to sleep, of which he formerly complained.

	mm.
Length of the hand from the lower fold of the wrist to the end of the middle finger	195
Length of the middle finger, starting from the palmar fold at its base	83
Length of the middle finger on the dorsal aspect, starting from the base of its first phalanx	100
Length of the little finger, palmar aspect	66
Circumference of the hand, without the thumb, at the head of the metacarpal bones	234
Width	95
Greatest circumference of the "obstetric hand"	280
Greatest thickness of the hand (with callipers) at the level of the thenar eminence	57
Circumference of the middle finger	85
Circumference of the thumb	90

	mm.
Circumference of the little finger	75
Circumference of the wrist, immediately below the extremities of the ulna and radius	200
Circumference of the wrist at the level of the styloid process of the ulna	198
Circumference of the forearm (at the middle)	278
Circumference of the arm (at the middle)	315
Length of the nail of middle finger	14
Length of the nail of the thumb	16
Breadth of the nail of middle finger	16
Breadth of the nail of the thumb	23
Length from the iliac crest to the summit of the head of the fibula, (the patient was in bed)	540
Length from the summit of the head of the fibula to the tip of external malleolus	370
Vertical diameter of the patella	66
Transverse	75
Circumference of thigh (at the middle)	515
Greatest circumference of the calf	400
Circumference immediately above the tip of the internal malleolus	287
Greatest length of foot	280
Circumference over heel and instep	395
Greatest circumference of the foot	265
Greatest width of foot	104
Circumference of the great toe	110
Circumference of the little toe	70
Length of the nail of great toe	19
Width of the nail of great toe	20
Length from top of forehead to tip of chin (with callipers)	280
Length from top of the forehead to the upper part of nasal bones	79
Length from the upper part of nasal bones to tip of nose	63
Greatest width of olæ nasi	35
Distance from the tip of the nose to the point of junction of the latter with the upper lip	28
Length from septum of the nose to the point of the chin	80
Greatest distance between outer surfaces of cheek bones	136
Width of mouth	55
Vertical measurement of lower lip	16
Transverse measurement of tongue at the middle	90
Thickness of tongue (at the middle)	190

	mm.
Length of one of the borders of the tongue, drawn out of the mouth, from the tip to the point where this border meets the upper lip	70
Lower jaw, vertical measurement from the free border of the gums to the lower part of the symphysis (with callipers)	44
Distance (with callipers) from the temporo-malar articulation to the lower part of the symphysis of chin	145
Distance between the two angles of the lower jaw (with callipers)	116
Ditto (with tape measure along the body of the bone passing in front of the symphysis)	240
Ears, greatest length	62
Ears, greatest breadth	32
Circumference of the thorax over nipple	1110
Circumference of neck between hyoid bone and upper part of thyroid cartilage	450

SYMPTOMATOLOGY.

The description of this patient gives a very correct idea of the usual appearance in acromegaly, and most of the morbid phenomena are sufficiently marked for this case to be regarded as nearly typical.

It will however be useful to trace a general sketch of the disease, insisting on those symptoms, the study of which present special interest, and to point out its course, its different nosographical and etiological characters, and the difficulties which may arise when it has to be distinguished from a certain number of affections.

What characterises it clinically and allows us to make its diagnosis at the first glance, is, as I have shown, the *truly remarkable hypertrophy of the extremities* (hands, feet, cephalic extremity). Certainly there are many other deformities of the most different organs in acromegaly, but from the semiological point of view none seems to me to have the value of this hypertrophy of the extremities. The *hands* are enormous, like battledores; however their general form is almost regular but stumpy (camard), their width being rather out of proportion to their length.

The fingers present the form called "sausage shaped;"

often there is manifest swelling of the articulation of the first and second phalanges (somewhat analogous to the nodosities of Bouchard), with a certain flattening of the finger in the antero-posterior direction. The palmar lines are extremely marked and bordered by enormous folds. The hypertrophy affects not only the skeleton, but in a very marked degree the soft parts also; this hypertrophy of the soft parts is especially developed at the level of the upper part of the hand and at the ulnar border of the latter; there is there, towards the internal part of the hypothenar eminence, a large mass of flesh, which is easily isolated from the fifth metacarpal bone. The nails are flattened, rather widened but short; often they seem too small in consequence of the increased size of the fingers; they are distinctly striated longitudinally, their lateral borders are sometimes curved upwards when the hand is examined with the palm resting on the table.

The wrist itself is generally a little increased in size, but to a less degree than the hand; it is more rarely that the forearm participates in the hypertrophy, and only quite in its lower part; the arm maintains its usual size—sometimes indeed it appears less large in consequence of the flaccidity of the tissues.

On the part of the lower limbs the same characters: the feet are enormous; on their external border the mass of tissue forms an enormous pad. The tendo Achillis may appear increased in size (Saucerotte). The malleoli are generally more or less increased in size; likewise, but to a smaller extent, the head of the fibula and the upper extremity of the tibia. Otherwise the size of the leg is not found much increased. The knees often appear prominent in consequence of the increase in size of the patella, and of the condyles of the femur. The diameter of the thigh is unchanged.

The cephalic extremity presents, too, an increase in bulk, especially marked in the prominent parts of the face. The cranium is but little altered in shape and size, or at least its alterations are not obvious; the face however appears elongated vertically. The forehead is usually rather low, with a very marked prominence of the orbital arches (due especially to the dilatation of the frontal sinuses). The eyelids are often elongated, sometimes thickened; their tarsal cartilages may be hypertrophied. The nose is manifestly increased in all its dimensions, it is enormous, and in several patients I have seen it take very distinctly the form pug-nosed. The cheeks are generally flattened and elongated. The cheek-bones rather prominent and bulky (not from hypertrophy of the malar bones, but from dilatation of the maxillary sinuses; this prominence of cheek-bones is moreover in part masked by the elongation of the face). The increase in size of the lower lip contributes greatly to give to the patients the remarkable physiognomy which enables them to be recognised at a distance and at the first glance; this lip is protuberant and strongly everted. The upper lip too may be a little thickened, but not in a manner comparable to what has occurred in the lower lip. The chin projects markedly downwards and forwards, it is large and massive; moreover, the lower jaw is altogether considerably increased in size, and as the upper jaw does not undergo the same modifications, a very marked degree of prognathism often ensues. In consequence of the exaggeration in size of the lower jaw, the whole face is found to have a considerable vertical measurement, and takes thus the form of an elongated oval. The teeth undergo no modification in size, but in consequence of the enlargement of the lower jaw they are here seen to be separated a little one from another. The tongue is of enormous dimensions, and in some cases its volume may be estimated at double that which it has in the normal condition, but its shape always remains perfectly regular; the increase in size takes place in all directions, less perhaps in length than in width and thickness. These modifications of the lips and the tongue sometimes impede the patient's articulation.

As for the ears, they do not present always the same characters: sometimes their dimensions are quite normal; in other patients on the contrary they are notably increased.

But it would be a grave error to think when these phenomena—remarkable it is true on the part of the extremities—have been pointed out, that a complete picture of the disease has been presented; far from it. We shall see indeed that almost all the tissues experience, more or less, marked modifications.

The condition of the spine should especially be described with care if one wishes to have an exact idea of the appearance of the patient, for it influences considerably his attitude. However little the affection be pronounced, there is very marked kyphosis of the upper part of the dorsal region; the patient's head is buried in the shoulders, and his "humpback" is often the occasion of more than one joke.

Pretty often too a certain degree of scoliosis may be determined, but the latter is always much less marked than the kyphosis; indeed, there may be present in the lumbar region a certain amount of lordosis, this appearing to be compensatory. I cannot enter into a detailed description of the vertebræ; suffice it to say that they are very much hypertrophied.

The neck is generally thick; I have already said how short it is, and this shortness of the neck coinciding with length of chin and kyphosis, it is not rare to see the chin of these patients resting on the anterior surface of the sternum.

For the thyroid body, I cannot say definitely what is its condition; all that I can affirm is that if it seems sometimes a little atrophied it is never absent.

The thorax presents equally special characters. Without insisting here on the increase in size of the clavicles, the sternum and the ribs, I may point out the enormous circumferential measurement of the thorax, the obliquity of the ribs, the development of their cartilages, whence arises sometimes an appearance analogous to that of the rachitic rosary; pretty often too the lower ribs are seen to be strongly forced outwards. The form of the thorax is very remarkable when the affection is well-marked; in fact, this part of the skeleton appears flattened laterally, and on the contrary prominent in the antero-posterior direction; the sternal region is very protuberant and very oblique from above down and from behind forwards; the xiphoid appendix is enormous and its free extremity projects above the level of the sternum.

When the patient is told to make a deep inspiration the forward movement of the lower part of the thorax is quite peculiar. In these individuals respiration seems to be especially diaphragmatic. Professor Erb found in a woman affected with acromegaly a zone of retrosternal dulness, not present in two patients whom I have examined since the publication of this author's paper. Professor Vertraeten however has confirmed the existence of this symptom, which tallies with the state of the thymus in the disease.

I do not wish to insist any further on the malformations presented by the skeleton, as they deserve to form by themselves the subject of a special work, and I shall limit myself, in concluding, to recall the somewhat massive appearance of the pelvis.

The joints are as a rule rather large, sometimes nodose; they are often the seat of cracklings, often also of pains which may be rather acute.

With regard to the muscles, although it is true that in the cachectic period of the affection they appear flaccid and shrunken, it may be quite otherwise in the early stages, and I can affirm that two of my patients, whose stature moreover was not above the average, had muscular strength far above the normal. In the man whose case I am now reporting, the muscular system is really very well developed. Erb has found that in these patients muscular excitability by minimum currents was considerably increased.

If we pass in review the different mechanisms, we discover that a certain number present something abnormal.

Among the phenomena of sensibility, the most notable symptom is headache, which is present in the greater number of cases (but not in all); it may be very intense, and two of my patients who had given but little attention to their deformity had come to receive medical treatment simply for their headache. I have spoken above of joint pains which may be observed. It must be added that in one female patient Erb has discovered slight sensory affections of the forearms and hands.

In the domain of the special senses, sight is most often and most manifestly affected; and when the disease is sufficiently advanced we observe complete blindness in consequence of compression of the optic nerves by the enlargement of the pituitary body; or else, in less-marked cases, there is only slight visual trouble, but it is already possible to find with the ophthalmoscope indications of optic neuritis.

Hearing may be equally affected; as for taste and smell, we know but little with regard to them.

The skin is generally flaccid, sometimes dry, most frequently presenting a yellow-brown discoloration, sometimes slightly olive and especially marked on the eyelids. Sometimes it is the seat of vergetures: two of my patients had a few pendulous growths of molluscum; it is possible that this is only a common lesion, not depending at all on the acromegaly. The hair and the beard, in all the cases which I have observed, were thick and coarse.

The larynx is generally increased in size, and probably as a result of that increased size, the voice is strong and generally very deep; in one of my patients its compass was from mi_0 to ut_3 (E to C_3).

From the point of view of the digestive apparatus, I shall point out the almost insatiable appetite observed in certain patients, and also the no less excessive thirst. These phenomena moreover are not constant. I have observed them several times, and other authors have also recorded them; they exist in the patient I am now describing but he is a diabetic. Must we attribute the polyphagia and polydipsia to the diabetes or to the acromegaly alone? I cannot say. Finally, is diabetes a usual complication of acromegaly? This again is a question which I cannot decide for want of evidence. I must limit myself now to noting these facts.

The same remarks apply to the quantity of urine which in some cases has been very abundant.

For the circulatory organs, I may mention among the modifications they present, the increase in size of the heart, which I believe is frequent, and the tendency to venous dilatations (varicose veins, hæmorrhoids), which are found more or less marked in most of the patients, if not in all.

The genital apparatus is no more exempt; the penis, which according to the very true remark of Erb, "is also an *ακρον*," has sometimes (Brigidi, Klebs and Fritsche, and my Spanish patient) dimensions above the normal, but not constantly. Most often there is in the man a diminution of desire and power which may reach to complete abolition. In the woman the most important phenomenon, on which moreover I have insisted in my first work, is the *suppression of the menses*, which is almost always an early phenomenon, so much so in most cases, that it may be considered an initial symptom and one from which the commencement of the disease may be dated. From the anatomical point of view, I may point out the increase in thickness of the soft parts of the external organs of generation (Erb), the unusual dimensions of the clitoris, the prepuce of which is thickened, the width of the vagina and of the posterior *cul-de-sac* (Freund). We can equally, in the woman, prove the absence of sexual desire.

The psychical functions are most often well preserved; sometimes indeed the good humour of the patients contrasts with their miserable condition; in other cases they give way to melancholy which may even lead them to suicide.

Such in its principal features is the clinical aspect of acromegaly. Its course is of very long duration—twenty, thirty years, and even more. The onset in the majority of cases seems to occur between the ages of twenty and twenty-six; but hitherto we have failed to obtain definite data on this point. Since the diagnosis is made only when the affection is very advanced, we have to trust entirely for the period of onset to the patient's statements. At the very commencement the symptoms are but little noticed, except the suppression of the menses or the headache. However, the dimensions of the extremities continually increasing, the patient is astonished to perceive that he has to change his fit as well for his shoes as for his gloves; some individuals do not even notice that they have become prognathous. Later on (but perhaps not always) arise affections of vision which sometimes end in complete blindness. Finally, little by little, the patient falls into a condition of progressive cachexia which necessitates his confinement to bed;

this lasts a few years, and then death supervenes in an unexpected way, with the indications of syncope.

Diagnosis.—It seems that an affection presenting such a group of quite special characters should not offer any difficulty from a diagnostic point of view; in reality it is not always so.

The affection described by Virchow[1] under the term *leontiasis ossea*, will hardly give rise to confusion, for here we are concerned with the development of true bony tumours on the face and the cranium, producing great deformity and a truly hideous appearance; in acromegaly, on the contrary, the bones of the face and of the cranium are the seat of a more uniformly distributed hyperostosis (or rather the increase in size is due much more to the dilatation of the frontal sinuses than to a true hyperostosis), without the formation of osseous tumours or definitely circumscribed bosses. Finally, leontiasis ossea is not associated with hypertrophy of the limbs.

I do not think it any more useful to insist at length on the differences which separate acromegaly from *elephantiasis*, the latter affection consisting in hypertrophy with œdema of the skin and of the subcutaneous areolar tissue without involvement of the skeleton; moreover, it is often unilateral and scarcely ever affects the upper limbs and the face. The aspect of the affected limbs is here completely modified, their contours are completely altered, they form only a shapeless mass; in acromegaly, on the contrary, the prominences and the contours of the limbs remain perfectly normal.

Another affection which is associated with an increased development of subcutaneous tissue, deserves to attract more attention—I mean *myxœdema*, and more than one case of acromegaly has been regarded and published as a case of myxœdema. To avoid this error however it is sufficient to remember that in myxœdema the dimensions of the skeleton are in no way changed, that although the extremities may appear swollen, they are not hypertrophied, and that the face has a characteristic form like a full moon

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[1] Virchow, 'Pathology of Tumours.'

(Sir William Gull), whilst in acromegaly the face is considerably elongated and of a very well-defined elliptic form.

There is another disease to which at first sight acromegaly may seem closely allied, although in reality it is quite distinct from it; it is that curious disease described for the first time in England, and in a very remarkable way, by Sir James Paget who has given it the name of osteitis deformans. In consequence of the ambiguity to which this name gives rise—having been already applied to other forms of bone changes of chronic course—I proposed, at the time when I was the first in France to make common the description of this affection hitherto unknown amongst us although several times observed, to give it the name of Paget's disease, at the same time noting that it would be necessary to avoid confusion with the other so-called "Paget's disease," that of the nipple. My proposal was well received, and now this affection is usually designated in France under the name of "*maladie osseuse de Paget.*" I think that this designation will be equally accepted in England as an appropriate one.

The points in which at first sight the *maladie osseuse de Paget* approaches acromegaly are, increase in size of the limbs and increase in size of the head. But if we examine the facts with a little more attention we shall soon be convinced that these analogies are only apparent; the distinctions on the contrary are considerable. Indeed in the bone disease of Paget it is especially the cranial bones which by their hyperostosis produce the increased size of the head; if sometimes the facial bones are themselves affected, it is only to a slight and so to speak accessory degree. In acromegaly on the contrary it is more especially the facial bones which undergo hyperostosis; also in the former the face takes on a triangular shape at the lower part, whilst in the latter it has that of an elongated ellipse, and we have seen that in myxœdema it is rounded "like a full moon," as Sir William Gull has very justly observed.

As for the localisation of the hyperostosis in the limbs, it is far from being alike in the two affections. We have seen that the special character presented by our patients is an enormous hypertrophy of the feet and of the hands, coming on most frequently without notable change in size of the long bones of the limbs, and, at least, long preceding the latter when it exists, whence comes a strange contrast between the width of the extremities and the slenderness of the limb itself. Now in Paget's disease it is quite otherwise; the long bones especially are affected, the hyperostosis scarcely ever involving the bones of the extremities, or when it does so it is only in a very slight degree.

In Paget's disease we perceive moreover a very marked tendency in the diaphysis of the long bones to undergo quite abnormal curvatures, whence the name "osteitis deformans;" except in the case of the spine there is nothing of the kind in acromegaly.

The onset also is quite different: the first of these diseases only manifests itself after the age of forty years; the second, on the contrary, almost always between twenty and thirty. Further, in the second, invasion of the different parts of the skeleton occurs symmetrically, that is to say, the two hands, the two feet at a time, whilst in osteitis deformans invasion occurs in a much more dissociated manner; one tibia or one femur is first attacked, the corresponding bone of the opposite limb becoming affected only after a certain time; and throughout the whole course of the disease the bones of the side first affected may be seen to be more hypertrophied and more deformed than those of the opposite side.

In certain forms of *rachitis* we see patients whose faces seem too large for their stature, and notice especially the prominence of the frontal bosses, of the nose and of the chin. The hands and the feet of these individuals are equally of exaggerated size, and up to a certain point the appearance which they present recalls that of acromegaly; but there the analogy ceases, and when we examine into details we soon meet with fundamental differences; moreover, in these individuals we find deformities of the diaphysis of the long bones which do not manifest themselves in acromegaly.

With *gigantism*, the diagnosis would perhaps at first not seem to require long discussion but nevertheless it is under this title or analagous titles (macrosomia), that several cases of acromegaly have been published; this depends on the fact that the individuals attacked by this disease are sometimes of great stature, and then the increased size of the face and of the extremities being associated with this great height give to the eye the impression of a truly supernatural development, whence a tendency to look upon these patients as "giants," whereas still taller people do not produce this impression. Whatever the explanation may be, in order to avoid the error it will suffice to remark that in gigantism the extremities are in proportion to the stature, that the face is not elongated, that the jaw especially presents neither the hypertrophy nor the prognathism so characteristic of acromegaly. Finally, passing in review the different symptoms proper to this latter, we shall see that they are altogether absent in gigantism.

We must speak now of another affection, the diagnosis of which as distinct from acromegaly, is attended by still greater difficulties. This affection bears no name, and that of the physician to whom we owe the description of the two patients attacked by it could be applied only with an additional designation; "Friedreich's disease" having already the right of a place in nosology. I shall therefore describe this affection by the name of the patients themselves, the brothers Hagner. Here is a *résumé* of the facts with which we are concerned. Friedreich had in 1867 the opportunity of studying two patients, the brothers Hagner, whose feet had begun to increase in size towards the age of eighteen; then the legs, as high as the knees, had become thicker and firmer; two years afterwards both hands began also to become more bulky. When seen by Friedreich, the feet and the hands presented an appearance like that of elephantiasis, but even a superficial examination made it apparent that the increase in size of these parts was produced by an increase in size of the bones. Moreover, certain bones of the skeleton participated in this increased size (clavicles, ribs, sternum, malar bones, &c.). We see here a most striking analogy with acromegaly. In my first work ('*Revue de Medecine,*' 1886), after much reflection

I considered the brothers Hagner as belonging certainly to acromegaly. Since then Professor Erb having had the opportunity of again seeing these patients, published in 1888 *(Deutsches Arch. f. Kl. Med.)* the results of this fresh examination, and thus a certain number of facts were stated precisely which had not been sufficiently mentioned in Friedreich's descriptions. Thanks to this further information, I must now reconsider my first opinion. I may say in fact that the brothers Hagner do not appear to me to be cases of acromegaly; at the same time admitting that I cannot say what they are. Perhaps we have here a hitherto undescribed affection and one which should be isolated from the distinct group of hypertrophies of the limbs. The arguments on which I rely to establish this separation are the following:—

(*a*) The lower jaw by no means presents the very characteristic malformation described in the other patients.

(*b*) There is no increase in size either of the nose, the lips, or the tongue.

(*c*) The xiphoid appendix is small.

(*d*) The neck is slender.

(*e*) The kyphosis is seated not in the cervical and upper dorsal regions, but in the lumbar and lower dorsal.

These are indeed very important distinctions. One might strictly maintain that we have to do with an incomplete form of limited acromegaly, localised only in the limbs and the trunk, and not involving the head or the neck. I confess that this explanation would rather tempt me, but on one condition, which is, that in the form of the limbs we should find exactly the same appearance as that invariably proved in all typical cases of acromegaly. Now there is nothing of the kind. Let the description of the hands and fingers of the brothers Hagner be read, let their appearance be examined in the figures given in Erb's work, and it will be seen what differences separate them from the ordinary type. Likewise for the lower limbs, this complete disappearance of all human shape in the legs—this appearance like elephantiasis, in a word—is by no means characteristic of acromegaly; far from it. In this latter, the contours of the limbs preserve on the contrary, as a rule, a perfect symmetry, as can be verified on the different figures which we have published; in no case does one see the deformity observed in the brothers Hagner. From all these considerations it must be concluded that the latter should not be included among the demonstrated cases of acromegaly, although in them the extremities of the limbs may certainly have undergone considerable hypertrophy. Indeed this condition of hypertrophy of one or more of the limbs should not suffice when the other characters are wanting and for my part I cannot definitely admit a condition of *partial acromegaly,* which according to Professor Virchow could be deduced from the generalised acromegaly. This method, apparently highly philosophical, tends to nothing less than to create regrettable confusion in clinical medicine. The affection which I have endeavoured to isolate and to describe is one disease autonom and of a well-defined type, and I cannot admit that it should be confused with those unilateral hypertrophies of the face or of the limbs from which in my first paper in the '*Revue de Medicine,*' I had carefully separated it. We know that these hypertrophies may manifest themselves after different types—sometimes a unilateral hypertrophy of the face; sometimes hypertrophy of one or several fingers, or of one foot. We may even see unilateral hypertrophy of the whole body, homonymous, or crossed (one side of the face and one arm, and the lower extremity of the opposite side). But in all these there is indeed nothing which resembles acromegaly. Most often we are concerned with a congenital malformation; moreover, nothing suggests the idea of a progressive affection—the hypertrophy is, so to speak, the sole phenomenon. Finally, this hypertrophy, if it is often localised in *one* extremity, does not attack several extremities in a special way simultaneously; for example, in the case of unilateral hypertrophy of the body. Let established terms therefore be preserved —macrodactylia, macropodia, unilateral macrosomia, &c.; but let us avoid comparing what is not clinically comparable by the thoughtless use of the term "partial acromegaly." That would cause a confusion which could not be otherwise than prejudicial in the study of an affection yet imperfectly known like acromegaly is.

As for the etiology of this disease, we must indeed confess that we have scarcely any precise data on this subject; in several patients however syphilis could be blamed. In every case I think I can affirm that *heredity* does not play any part; acromegaly is not a family disease; it is not hereditarily transmitted.

Its frequency seems to be about the same in the two sexes.

At the end of this article will be found bibliographical references to the different cases of this affection which have come to my knowledge.

I should have wished in conclusion to pass in review the anatomo-pathological characters of acromegaly, but the study of this is very little advanced yet; for my own part I have had the opportunity of making but one single autopsy. The results of the examination of the skeleton of my patient have been published by my friend Dr. Auguste Broca[1]; this investigation has shewn us that it is especially the spongy tissue (short bones, flat bones, epiphyses) which is the seat of the hypertrophic process, so that the following statement may be considered as representing the reality; "in the skeleton of limbs from cases of acromegaly, hypertrophy shews itself in preference in the bones of the extremities, and in the extremities of the bones."

Independently of the bones of the limbs, I may point out the considerable hypertrophy of the vertebræ, the sternum and the clavicles. The frontal sinuses are the seat of a very well-marked dilatation. Finally, amongst the lesions affecting other organs, and which after what has been observed in other autopsies seem to me to be constant in acromegaly, must be mentioned hypertrophy of the pituitary body with enormous dilatation of the Sella turcica, persistence of the thymus, and finally hypertrophy of the cord and ganglia of the sympathetic system. Until proof to the contrary is brought forward I shall cling to the belief that these last three anatomo-pathological characters manifest themselves not only with a remarkable degree of frequency, but may even be looked upon as constant. The autopsies hitherto published in which these lesions have not been seen, were not of patients suffering of true acromegaly. The clinical picture offered by these cases was certainly different from that observed in the instances which I consider to be typical, and I feel absolutely certain that we have to do here with affections quite distinct from acromegalia.

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[1] Broca, *Archives générales de Medecine*, Dec., 1888.

RATIONAL BIBLIOGRAPHICAL INDEX OF OBSERVATIONS ON THIS DISEASE WHICH HAVE SO FAR COME UNDER MY NOTICE.

A.—Cases which may be considered with certainty as belonging to Acromegaly.

V. BRIGIDI.—Studii anatomopatologici Sopra un nomo divenuto stranamente deforme per cronica infermistié (Societa medico-fisica fiorentina.) Communicated 26 Aug. 1877.

W. ERB.—Ueber Akromegalie (Krankhaften Riesenwuchs) Deutsches Archiv. f. Klin. Med. 1888. T. lxii., fasc. iv , p. 296.

FARGE.—This case is still unpublished. It will appear in June or July 1889 in the *Progrès Medical.* The author is very desirous that I should communicate his manuscript and photographs.

W. A. FREUND.—Ueber Akromegalie. Sammlung Klinischer Vortrage von R. von Volkmann, 1889. Nos. 329, 330.

FRITSCHE ET KLEBS.—Ein Beitrag zur Pathologie des Riesenwuchses. Klinische und pathologisch-anatomische Untersuchungen, Leipzig, 1884.

RICKMAN J. GODLEE.—A case of acromegaly. Clinical Society of London, April 13th, 1888.

W. B. HADDEN AND CH. BALLANCE.—A case of hypertrophy of the subcutaneous tissues of the face, hands and feet, exhibited January 23, 1885—*Clinical Society's Transact.*, vol. xviii. A continuation of their observations on the same disease under the title: "A Case of Acromegaly," read April 13, 1888—*Clinical Society's Transact.*, vol. xxi.

H. HENROT.—Notes de Clinique Medicale, Reims, 1877; and Notes de Clinique Medicale, des lesions anatomiques et de la nature du myxœdeme, Reims, 1882.

LANCEREAUX.—Anatomie Pathologique T. III., 1ere partie, p. 29. Treats of a case of Basedow's disease, with deformation of the skull. In reading this description it appeared to me that the case was one, not of exophthalmic goître, but of acromegaly.

I expressed my doubts to M. Lancereaux and that eminent master graciously sent me all the notes he had preserved about the case. These notes which I expect to publish in treating of the pathological anatomy of acromegaly, show very clearly that the case in question is to be referred to that disease.

CES. LOMBROSO.—Caso singulare di macrosomia. Published at first in the Giornale ital. delle mallattie veneree, &c., 1868, translated by M. Fraenkel in Virchow's Archiv. T. xlvi., p. 253. Republished with considerations on partial osseous hypertrophy in Annali Universali di Medicina, T. ccxxvii., p. 505 et seq.

P. MARIE.—Sur deux cas d'acromégalie, Revue de Médecine, Avril, 1886, 2 cas. L'Acromégalie, Nouvelle Iconographie photographique de la Salpetrière. This second work contains only one new case (No. 1). No. 2, which I had considered as an example of this disease does not apparently belong to it. L'Acromégalie, étude clinique, *Progrès Medical*, Mars, 1889.

O. MINKOWSKI.—Ueber einen Fall von Akromegalie. Berliner Klinische Wochenschr, 1887. No. 21.

SAUCEROTTE.—Mélanges de Chirurgie, première partie, 1801, p. 407 et seq. Case read before the Academy of Surgeons in 1772.

CES. TARUFFI. — Della macrosomia. Annali Universali di Medicina, 1879. T. ccxlvii et ccxlix.

A. VERGA.—Caso singolare di prosopectasia in Rendiconti del Reale Istituto di Scienze e Lettere. Adunanza del 28 Aprile, 1864.

VERSTRAETEN. — L'Acromégalie. Revue de Médecine, May, 1889. This work contains two cases, only the second of which however concerns us here. As to the first, it will be referred to under Section C.

WADSWORTH.—A case of myxœdema with atrophy of the optic nerves—*Boston Medical and Surgical Journal*, Jan. 1st, 1885. It is to Messrs. Hadden and Ballance that we owe the recognition of this case of acromegaly, which, considered as myxœdema by the author, had previously passed unnoticed.

WILKS.—Clinical Society of London, April 13, 1888.

B.—Cases of which Details are wanting, but which very probably belong to Acromegaly.

ALIBERT.—Précis théorique et pratique des maladies de la peau. Paris, 1822. T. iii., p. 317.

W. O. CHALK.—Partial dislocation of the lower jaw from an enlarged tongue—*Transact. of the Patholog. Soc. of London*, 1857, T. viii., p. 305. It was M. A. Broca who first made this case known, and showed that, according to all appearance, it belongs to acromegaly.

Fred. Tresilian.—A case of Myxœdema.—*British Medical Journal*, March 24, 1888, p. 642. This case was brought to my notice by Professor Verstraeten.

Rud. Virchow.—Ein Fall und ein Skelet von Akromegalie. Lecture delivered before the Berlin Medical Society 16 Jan. 1889. Berliner Klin. Wochenschr., 4 Februar, 1889. No. 5.

C.—Cases in which the Clinical Aspect differs more or less notably from that of the typical cases of Acromegaly. These probably do not come under the head of Acromegaly at all.

Aug. Bier.—Ein Fall von Akromegalie, Mittheilungen aus der chirurgischen Klinik zu Kiel iv., 1888.

O. Fraentzel.—Ueber Akromegalie. Read before the Congrès de Médecin Interne—in Deutsche Med. Wochenschr., 9 Aug., 1888.

Friedreich.—Hyperostose des gesammten Skelets. Virchow's Archiv., Bd. 43, p. 83, 1868. Additional details about these two cases are given in the memoir of Erb, quoted above.

P. Marie.—L'Acromégalie. Nouvelle Iconographie Photographique de la Salpetrière, 1888. I at first believed the second case here described to be acromegaly, but doubts have since arisen about it in my mind, which are far from being dissipated.

Saundby.—This case was published in the *Illustrated Medical News*, 1889. I had no knowledge of it myself, but the author was kind enough to send me some microscopic preparations and a photograph of the patient. From this last I should be inclined to believe that the case was one analagous to that of the brothers Hagner (Friedreich).

Verstraeten.—L'Acromégalie, *Revue de Médicine*, Mai, 1889. It is the first case in this paper that falls to be considered here. In reading it attentively one sees that it differs very notably from the clinical aspect furnished by the typical cases. I am opinion therefore that it ought to rank in this third category must confess that at first, on looking at a photograph o patient sent to me by Dr. Verstraeten, I did think that it true case of acromegaly; but when I read the detailed de of the symptoms in the *Revue de Mèdicine* doubts ar mind about the correctness of this diagnosis.

THE PATHOLOGY OF SENSORY APHASIA, WITH AN ANALYSIS OF FIFTY CASES IN WHICH BROCA'S CENTRE WAS NOT DISEASED.

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In questions regarding the localisation of cerebral functions, the final appeal must always be made to pathology. Even as regards those functions which are common to man and brutes, such as motion, sight, hearing, &c., the conclusions reached by physiological experimentation require confirmation before they can be accepted as true of man. And when it is a question of the functions belonging to man alone, the pathological method is the only one. And, in fact, the data furnished by pathology prove that it is not safe to rely for exact localisation of human functions upon physiological results; for the fact that each species varies somewhat in the extent and limits of its functional areas is now pretty generally accepted.

While therefore the clinical observer cheerfully admits his debt to the physiologist for the discovery of the principle of localisation, and for the mass of evidence upon which that principle rests, he must rely for his exact data, as regards the application of the principle to man, upon the clinico-pathological method. This is especially true of sensory disturbances. And since it is the practical application of the principle to the removal of disease from the human brain which is the chief object in view, the paramount importance of pathological facts cannot be denied. The collection and analysis of reliable and well-observed ses of disease in man is therefore of value, and is the cessary preliminary to surgical therapeutic procedure.

In the study of aphasia and in the localisation of the various functions which take part in the use of language, clinical observation has always outrun pathological data. The loss of speech had been recognised clinically long before Broca showed the frequency of lesions in the third frontal convolution of the left hemisphere as its underlying condition; and English physicians had described and accurately differentiated the two great varieties of aphasia—ataxic and amnesic, or, as they are now generally known, motor and sensory aphasia—some years before Wernicke recorded the first cases with autopsies, which proved that the latter, in distinction from the former, had its own particular lesion in the first temporal gyrus.

Of late years, owing to the labours of Lichtheim, Ross, Charcot and others, the clinical varieties of aphasia have increased in number. Four very distinct forms have been recognised, numerous pure examples of each having been recorded by careful observers. These are motor aphasia, agraphia, word deafness and word blindness.

The recognition of the various elements making up the mental image of a word, all of which are necessary to its use, has resulted from a study of these varieties of aphasia, and the necessity of a physical basis for the association of ideas, for the union of these different elements making up the word image, has followed at once upon the discovery of the separate situation of the memory pictures of sight, sound, and manual or vocal effort involved in the word image. It has become evident that a division of association tracts as well as a destruction of memory pictures may give rise to disturbances of speech. And already attempts are being made to differentiate clinically the forms of aphasia due to disturbance in association of ideas, from those due to a destruction of the elements making up the idea.

But here clinical observation has far outrun pathological facts. It seems necessary therefore to ascertain to just what degree the pathological data give support to these clinical distinctions. What is the present status of the pathology of aphasia?

It will be admitted that no doubt exists at present in regard to the pathology of motor aphasia. Facts in accord with the dictum of Broca, that a lesion of the posterior part of the third frontal convolution on the left side in right-handed and on the right side in left-handed persons produces a loss of the power of using language without any disturbance in the power of understanding words, are too well known and too numerous to require more than a simple statement. And the additional fact that the same effect, though usually but temporary, may follow destruction of the motor speech tract from Broca's centre to the motor nuclei of the pons and medulla rests upon positive data.[1] In the latter case correlated symptoms may enable the position of the lesion to be recognised during life, these symptoms being such as are usually produced by foci of disease in the internal capsule, crus cerebri, or pons varolii.

In regard to the pathology of sensory aphasia, the number of positive facts is by no means large. The pathology of sensory aphasia rests much more upon forcible assertion and reiteration, and upon the analysis of ingenious diagrams, than it does upon the collation of reliable evidence. One or two cases, supported by a very few others, have formed the basis for many far-reaching statements; and there is by no means the certainty regarding the lesions in sensory aphasia which is desirable.

In 1874 Wernicke[2] collected ten cases of sensory aphasia with lesions, and drew from them a brilliant conclusion which has been confirmed by subsequent observation.

In 1884 Seppilli[3] went over the subject and brought together seventeen cases, discarding however all but two of Wernicke's cases as not without objection.

In 1885 Amidon,[4] in presenting Seppilli's article in English, added four cases from American literature. In 1887 Naunyn[5] in a general survey of the pathology, collected in all ninety cases of aphasia, but twenty-four of which can be regarded as applicable to the solution of questions arising in regard to sensory aphasia, since in all the remainder Broca's centre was injured in some degree. A careful search through medical literature of the past twenty years has resulted in a collection of fifty cases of aphasia of a distinctly sensory variety, which may be utilised for conclusions, and which have been tabulated. But in the collection of these cases a larger number has been found which had to be excluded from the table. For it is evident that in the selection of cases for conclusions certain criteria must be applied. It was thought best to exclude all cases in which the lesion had invaded Broca's centre, or had encroached extensively upon the Island of Reil. For in such cases it is practically impossible to satisfy one's self as to the extent to which the symptoms may be ascribed to the motor disturbance in the use of words. It was also thought that conclusions would be unreliable if derived from cases in which death had followed very soon after the onset of the symptoms, since in many other cases a considerable modification of the symptoms occurred after the first few weeks. And lastly cases were excluded in which the nature of the disease—*e.g.*, large tumours, threw doubt upon the separation of indirect from direct local symptoms, *i.e.*, prevented a sharp line from being drawn between the effects of general increase of intracranial pressure and effects due to a destruction of a limited zone of tissue. All but thirteen of these cases have been observed within the past decade, and hence careful clinical examination and accuracy of pathological description has been secured in these, while the thirteen selected have conformed in this respect to necessary requirements. The cases are given in the order of their publication, being numbered. A table is then appended containing a pathological and clinical analysis, thus bringing into view at once the lesion and symptoms, and enabling a comparison of cases to be made. And finally, the conclusions which may be legitimately drawn from this material are presented.

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[1] Raymond et Artaud, 'Atch. de Neurol., 1883,' No. 20.

[2] 'Die Aphasische Symptomen Complex,' Breslau.

[3] 'Revisita Sperimentali,' 1884.

[4] 'New York Medical Journal,' Feb. 1885.

[5] 'Verhandl d. Cong. f. Inn. Med.,' Wiesbaden, 1887.

Conclusions :—

(1.) In all of these cases some form of sensory aphasia was present, and in all the lesion lay in the posterior lower third of the brain. The convolutions were found affected in the following order:—

First temporal	in 38 cases.
Second ,, ...	,, 27 ,,
Inferior parietal	,, 21 ,,
Angular gyrus	,, 25 ,,
Supra marginal gyrus	,, 12 ,,
Occipital lobe	,, 19 ,,

In seven of the cases pure word deafness was present. The patients had lost the power to understand speech when heard, though able to read, to talk and to write (Cases III., XVIII., XX., XXX., XXXIII., XXXIV., XL.).

In all of these cases the lesion was limited to the first and second temporal convolutions in their posterior two-thirds.

In eleven of the cases pure word blindness was present. The patients had lost the power to understand words when seen, though able to understand speech and to talk (Cases II., XXIII., XXXV., XXXVI., XXXVII., XXXIX., XLII., XLIII., XLIV., XLVI., XLVII.).

In two of these cases the patients were able to write or copy, but in the remainder they had lost the power or were not tested.

In these cases the lesion was not found uniformly in one location. It affected the angular gyrus in five cases, the occipital lobe in five cases, the temporal convolutions in three cases, the inferior parietal region in three cases, and the supra marginal gyrus in two cases.

By inferior parietal convolutions it is intended to indicate those gyri which lie between the supra marginal gyrus and the angular gyrus, and which are between the inter-parietal sulcus and the first temporal sulcus, the area lying between P 2 and P 2′ in Ecker's diagram, reproduced in "Ferrier's Functions of the Brain," p. 472.

In twenty-five of the cases the power to recall words and to name objects was impaired. This occurred in some of the cases of pure word deafness and also in some of pure word blindness. In some of these cases the power to recognise the word or name of the object when suggested by another person was preserved. And the lesion in these various cases varied widely, involving any or all of the various gyri included in the sensory speech area, or the subcortical tracts beneath them.

In seven of the cases word deafness and word blindness were present together, and yet the use of language was not lost. The patients could talk (Cases I., VII., XV., XIX., XXIV., XXIX., XXXIV.).

In these cases the lesion lay in the temporal convolutions alone in two cases, and in the remainder it extended posteriorly, involving the inferior parietal, angular and occipital convolutions.

In twenty-seven of the cases word deafness and word blindness were accompanied by more or less impairment in the power to talk. The difficulty in talking in but two cases was a difficulty in the power of pronunciation, such as occurs from lesion of Broca's centre. In all others it consisted of a use of wrong words, or unintelligent phrases, a series of words whose connection was deficient. Paraphasia is therefore the usual accompaniment of sensory aphasia. In these cases the lesion was wide in extent, involving the temporal, parietal and occipital convolutions.

It was impossible to ascertain any constant pathological difference between the cases of sensory aphasia without and with paraphasia. Nor did the power to repeat words one after another seem to depend upon the relative situation of the lesion, as might be supposed from Wernicke's assertion that this defect appears with paraphasia when the temporo-frontal tract is involved. For paraphasia with inability to repeat words was found in a few cases where the lesion lay too far back to affect this tract. Paraphasia therefore may be caused by lesions in very various locations.

The analysis of the pathological lesions therefore, does not bring out as clear a differentiation of the different forms of aphasia as might be desired.

It is evident that word deafness is due to a lesion of the first and second temporal convolutions. It is evident that word blindness may be produced by lesions lying in the region of the inferior parietal lobule, or extending either anteriorly from it into the temporal region or posteriorly into the angular gyrus and occipital lobe. It is evident that

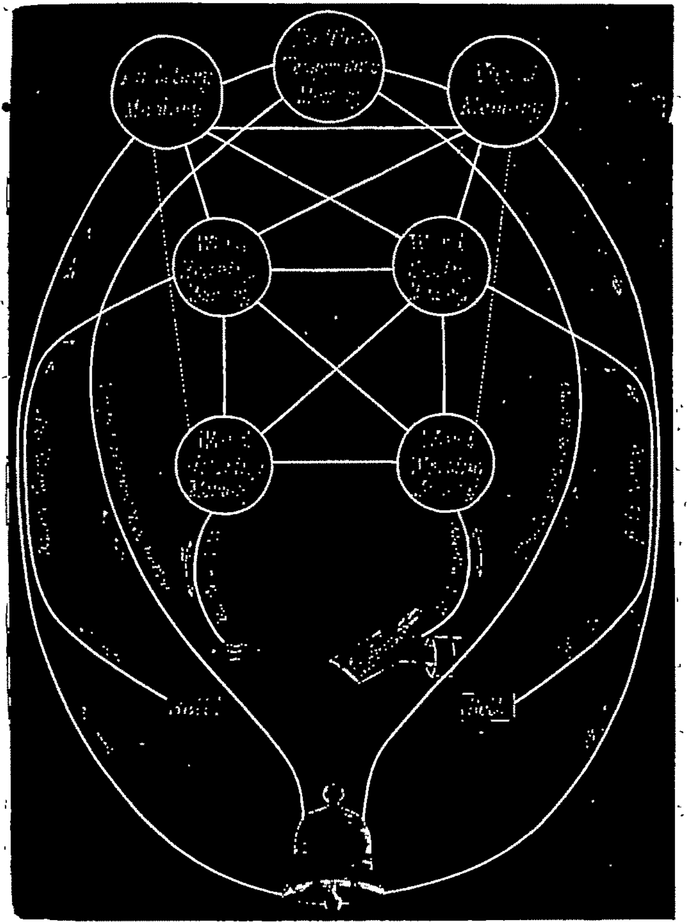

FIG. 1.—DIAGRAM TO ILLUSTRATE APHASIA.
THE CONCEPT "BELL", (modified from Charcot).

The mental-image of the bell is made up of auditory, tactile and visual memories joined together by association. To this is added the word-image "bell" made up of the memories of the word as heard and seen, as spoken and written. Each of these memories is associated with the others, and with the memories forming the mental image. All these memories together form the concept. Such a concept has no single location. It may be destroyed by general cortical disease (as in paretic dementia). It may be impaired in parts (as in psychical blindness or some form of aphasia). It may be seriously impaired by lesions involving the association fibres. The centripetal tracts from eye and ear, and the centrifugal tracts to mouth and hand, are also shown in the diagram.

these conditions are usually associated, and when occurring together are usually accompanied by paraphasia; in these cases the lesion may lie anywhere within the limits of the sensory aphasic area, which includes the inferior parietal convolutions, the two temporal convolutions and the occipital convolutions.

(2.) Approaching the subject now from the psychological and clinical side, let us see whether these cases establish, in any degree, some of the hypotheses which have been reached regarding the physical basis of speech.

If the process involved in recalling the name of an object be considered, it becomes evident at once that it is not a simple one. The concept of the object in the first place must be brought to mind, and this is made up of the various residua of perceptions by various senses.

Thus, of the object a bell, to use Charcot's illustration, the mental image includes that of the tone of the bell, of its appearance, and of its cold, hard feel. Granting that these residua of perceptions are present to the mind, to call the name into consciousness requires an association of the residuum of the word "bell" as heard with one or all of these parts of the concept.

To name the bell on seeing it is to revive one association; to name it on hearing its ring is to call up another association; and to tell the name of an object felt but not seen or heard, requires the passage of an impulse along still a third tract.[1] It is rarely that in the examination of an aphasic these various processes of association are separately tested. And therefore the statement that a patient is unable to recall words has a very indefinite significance, and can be only taken to imply generally that the processes of association are impaired. More care should therefore be taken in future in the examination of such patients, and these various processes must be tested separately. Thus granting for the moment that the memory of the name of an animal, say a dog, is located in the temporal region, and that the olfactory and visual memories are located respectively in the uncinate gyrus and cuneate gyrus, it becomes evident that to name the animal on seeing him, and to name him on smelling him, involves associations in exactly opposite directions; the former along the visual-auditory tract from behind forward in the temporo-occipital lobe; the latter along the olfactory-auditory tract, from before backward in the temporo-sphenoidal lobe.

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[1] I have a patient at present under observation who, though able to name things seen or heard, cannot give the name when the object is felt. She also has word blindness, and right hemianopsia.

The majority of patients are tested by showing them objects and asking them to tell the names. This then, is a test of the visual-auditory tract, between the occipital and temporal lobes of the brain. Such a tract, according to Meynert,[1] lies beneath the cortex covering in the white matter between these areas, a region which must be termed temporo-parieto-occipital, since the gyri of all three lobes lie upon it. Hence lesions of the annectant gyri and angular convolution, also those of the inferior parietal lobule, are very likely, if at all deep, to involve this tract.

It is not surprising to find that in the cases here brought together the power of recalling the names of objects was impaired or lost in a large number, and that the lesions producing this effect are by no means uniform in position; some lying forward in the tract within the temporal region, others about its middle in the parietal region and in the angular gyrus, others far back in the occipital lobe exclusively. The loss of power to recall the name of objects seen does not therefore indicate very exactly the seat of the disease.

But there are certain additional tests which aid in a more exact localisation of verbal amnesia. If the name cannot be recalled because the memory of it is lost, that is, because the physical basis of that memory has been not merely isolated from one connection, but actually destroyed, then it is evident that no other association will suffice to reach it, and that even recognition is impossible.[2] This is the condition in absolute word deafness, which as we have seen, is due to a lesion in the posterior two-thirds of the first and second temporal convolutions. To fail to recall a word by any means, and to fail to recognise its meaning when heard, is therefore characteristic of a lesion of this area when that lesion is extensive.

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[1] "Psychiatrie," p. 40.

[2] Attention was first called to this fact by De Watteville, 'Progrès Medical,' March, 1885.

But there are cases on record where the power to recall words is impaired while the power to recognise them remains. Cases II., VIII., XXII., XXXVII., XLII., XLIII., XLIV., XLVII., illustrate this condition. This must imply that the auditory memories remain and can be reached through the auditory tract, while the association fibres alone are affected by disease. Now the lesion in all of those cases lay in the posterior portion of the sensory aphasic area, the temporal convolutions being invaded in only two cases, and in those, only at the extreme posterior portion; while in all the inferior parietal convolutions and angular gyrus or occipital lobe were affected. And in all of these cases the lesion was a deep one, invading the white tracts beneath the convolutions; in five of them the lesion was wholly subcortical (Cases II., XXII., XXXVII., XLII., XLVII.).

The conclusion therefore appears to be warranted that while failure to recognize a word heard implies destruction of the temporal cortical area, failure to recall the name of an object seen implies destruction of the temporo-occipital association tract in the subcortical white matter.

The latter with the former implies extensive cortical and subcortical disease; the latter without the former implies subcortical disease only, without reference to its extent.[1]

There is a third condition which cannot be passed by in this connection, viz., psychical blindness. If an object is seen but not recognised, it implies that the visual mental image of the object is either destroyed or wholly cut off from its associations. This occurs not infrequently in lesions in the occipital lobe, either unilateral or bilateral. It is very frequently accompanied by the characteristic symptom of a lesion of the occipital lobe, namely, bilateral homonymous hemianopsia.

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[1] An interesting clinical example of a lesion in the association tract alone is given by Hughes Bennett in the 'British Medical Journal,' 1888, i. p. 339, case third.

In twelve of the cases here collected psychical blindness was present. In six of these hemianopsia was also present. In all these cases the occipital lobe was diseased; twice with the adjacent angular gyrus. Psychical blindness is produced not only by disease in the cortex of the occipital convolutions, but also by disease in the white tracts within the lobe. It is evident therefore that the memory pictures of objects lie in the occipital lobe, and a serious loss of them implies a lesion in that region. If the lesion be extensive enough to involve the cuneus, or deep enough to reach the visual tract to the cuneus as it passes beneath the angular gyrus and convexity of the occipital lobe, it will produce hemianopsia. If not, actual blindness may not accompany psychical blindness. In either case it is found that when things are not recognised, they cannot be named when seen. Hence the symptom of psychical blindness may aid in locating a lesion in the visual-auditory tract, and indicates that the lesion of that tract is in its posterior portion.

We thus have three sets of symptoms which enable us to locate a lesion in the association tract between the occipital and temporal areas, viz., loss of power to recognise the name of an object when heard (word deafness); loss of power to recall the name of an object recognised (verbal amnesia); loss of power to recognise an object seen whose name is understood (psychical blindness). The first implies a lesion in the temporal end of the tract. The last implies a lesion in the occipital end of the tract. The second implies a lesion between the others, probably beneath the inferior parietal lobule. And reference to the cases cited shows that the clinical distinction is supported by the pathological finding; that the psychological hypothesis has a confirmation from the facts of disease.

(3.) The principle applied to the study of lesions in the visual-auditory tract may be extended to the consideration of other tracts. If word blindness be held, as it justly may, to be merely a variety of psychical blindness, it becomes evident that a distinction must be made between recognising printed words, recalling printed words, reading aloud, or writing. Hence varieties in the condition of word blindness are possible.

To recognise the meaning of a sign implies integrity in the perceptive process and integrity in the associative process which joins the memory of that sign to some other mental image, giving it meaning. It is the association of two mental images which lies at the basis of any process of understanding. When we see the word "bell," it has a meaning only because the image of the word is associated with that of the object, and a destruction in the process of association will impair at once the power of recognition. It is not surprising, when this is understood, to find that the lesions producing word blindness are situated in various regions. When limited in extent and strictly cortical, the lesion producing word blindness was found in five cases in the angular gyrus and in the cortex immediately anterior to this in the inferior parietal lobule (Cases XXIII., XXXV., XXXIX., XLI., XLVI.). It is here therefore that the visual memory pictures are thought to lie. And in fact, in all the cases in this collection in which the lesion involved this area and in which reading was tested, there was word blindness (twenty-one cases).

The associations between the memory of words seen and the mental image are very numerous, and reach out in different directions. The words *thorn*, *trumpet*, *Madonna*, call up painful, auditory and visual memories respectively, and one can readily imagine that each of their associations might be impaired without the others being affected. But in all three cases this impairment of association might manifest itself as word blindness. Granting the hypothesis then that a lesion of the association tracts will produce word blindness,[1] it is evident that tracts going out in all directions from the angular gyrus as a centre might be invaded, with the result of producing the same symptom. The two chief tracts, those usually tested, will be those to the visual area and to the auditory area. To recall the appearance of an object on seeing its name, and to pronounce the name on seeing the word, are the tests applied to these tracts respectively.

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[1] This hypothesis was first broached by De Watteville, 'Prog. Medical,' March, 1885. Freund appears to have overlooked this fact in a recent article 'Arch. f. Psych. XX., Uber Optische Aphasie und Seelenblindheit."

Reading understandingly and reading aloud are therefore different processes, the former testing a tract from the angular gyrus backward to the occipital region; the latter testing a tract from the angular gyrus forward to the temporal region. A loss of the power to read may be associated with lesions in the occipital lobe, as in Cases XXXVII., XLII., XLIII., XLIV., in which the lesion was confined to this region. It may also accompany lesions in the posterior temporal region, which encroach upon the inferior parietal lobule, as in Cases II., X., XII., XXI., XXXVI.—in all of which, though the angular gyrus was intact, the symptom was produced.

The combination of word blindness with word deafness, in temporo-parietal lesions, and the combination of word blindness with psychical blindness and hemianopsia in parieto-occipital lesions appears to be established. Berlin's[1] condition of dyslexie, in which a patient is fatigued unduly by reading, and which he ascribes to a subcortical lesion beneath the angular gyrus, might well be explained by an interference with the association tracts.

Another tract which may be tested in this connection is the tract from the angular gyrus to Broca's centre. It is tested by asking a patient to read aloud, a process which may be gone through even though the patient does not understand what is read, as in Cases VII. and XXIII.[2]

Whether this tract is a direct one, or is indirect, viâ the temporal lobe, is still undecided; and sufficient material is wanting for definite conclusions, though in one of the cases here cited, reading aloud was possible where the temporal lobe was so much injured that word deafness was present. This case would indicate that the tract is a direct one, and if so, it must pass beneath the Island of Reil from behind forward. That it starts from the angular gyrus and passes forward into the inferior parietal lobule and supra marginal convolution is evident from the fact that in all the cases here recorded in which reading aloud was impossible, these parts were invaded by disease.

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[1] Berlin, 'Eine Besondere Art von Wortblindheit,' Wiesbaden, 1887.

[2] A similar case is reported by Hughes Bennett, l. c.

The last tracts to be tested in connection with printed language are those concerned in writing. Writing spontaneously and copying appear to test the same tract.

In all cases in which these powers were both tested, they were both lost or impaired equally. In all these cases the lesion was in or very near the angular gyrus. This tract starts then from this centre. Its direction and termination are not, however, known, as there are no autopsies upon cases of pure agraphia, excepting in the case of Sigaud (Case XLI.), where the lesion was confined to the angular gyrus and the condition was one of sensory rather than of motor agraphia.

It is evident from this review of the clinical and pathological facts in sensory aphasia that the pathological data warrant a recognition of many of the numerous forms of aphasia recently described. There are aphasias of association as well as cortical aphasias. It is necessary to recognise aphasia from lesion of the visual-auditory or occipito-temporal tract (verbal or auditory amnesia); aphasia from lesions within the occipital lobe giving rise to word blindness with visual amnesia; aphasia from lesions in the tempora-parietal region giving rise to word blindness with word deafness; as well as the simpler forms of cortical aphasia known as word deafness, word blindness, agraphia and motor aphasia. Thus far the clinical facts rest on pathological findings. Subjective investigation of speech processes, as well as clinical observation, may warrant further distinctions not yet resting on post-mortem records. It is evident that the various possible mental processes involved in memory must be carefully tested in every case; and that small subcortical lesions should not be overlooked.

Inasmuch as this investigation of the pathology of sensory aphasia shows the need of more careful examination of aphasics, it may be well to suggest the lines along which such an examination should be made.

It is necessary to investigate:—

1. The power to recognise objects seen, heard, felt, smelt or tasted.

2. The power to recall the names of such objects.

3. The power to recognise the names of such objects when heard.

4. The power to call to mind the objects when named.

5. The power to understand speech.

This examination will test the various sensory areas, and especially the temporal convolutions and the association tracts between these convolutions and the different sensory areas. It is also necessary to investigate:—

6. The power to understand printed or written words.

7. The power to read aloud and to understand what is read.

8. The power to recall objects whose names are seen.

9. The power to write spontaneously, and to write the names of objects seen, heard, &c.

10. The power to copy and to write at dictation.

11. The power to read understandingly what has been written.

These tests will determine the condition of the visual word memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas. It is necessary to find out whether:—

12. The power to speak voluntarily is preserved, and if not, the character of its defects.

13. The power of repeating words after another should also be tested.

The practical application of the localisation of lesions in aphasia hardly requires more than a mention. It is obvious. The regions of the brain concerned in speech are especially accessible to the surgeon, and experience has shown that subcortical tumours and abscesses are as open to operation as cortical lesions.[1] It is evident that in cases of sensory aphasia the trephine should be applied, not over Broca's centre in the frontal region, but over the temporo-parietal region; in word deafness over the posterior temporal region; in word blindness over the angular gyrus; in both combined over the inferior-parietal region, especially if verbal amnesia is present.

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[1] See cases of Seguin and Weir, 'Amer. Jour. Med. Sci.,' 1888; Ferrier and Horsley, 'Brit. Med. Jour.,' 1888, i. p. 530; Roswell Park, 'Trans. Cong. Amer Phys. and Surg.,' 1888.

The probability of a lesion lying anterior or posterior to the inferior parietal region may perhaps be determined by applying the tests for verbal amnesia already discussed.

(4.) It will be noticed that the results of pathological observation fail to give any support to the hypothesis of an "ideational centre" which Broadbent and Kussmaul have introduced into their diagrams.

Introspection will convince any one that thought is conducted either by the use of language or by the use of mental images of a definite sensory kind. Numerous distinct images combine to form the simple idea or "recept," to adopt the excellent term proposed by Romanes,[1] and the facts of psychical blindness, psychical deafness, &c., teach that this recept may be destroyed in parts by single lesions which invade various areas, obliterating distinct memory pictures, but that it is never destroyed in its entirety by a single cortical lesion. The facts here collected also demonstrate that subcortical lesions destroying the association tracts, whose integrity is needed to associate the various memory pictures into a recept, may impair that recept. And it is interesting to notice that these association-tracts interlace most freely under the temporo-occipital annectant gyri, where a lesion produces much mental confusion. To the recept of the object is added the recept of the word, and together, these make up the simple concept. But in addition to these numerous and various memory pictures associated together there seems to be no reason to hypothecate the existence of an idea, or to suppose any ideational centre, and the facts of pathology support this view. Ideas higher than simple concepts require language for their use, and are impaired when aphasia is present. Thought being regarded as the play of consciousness along lines of association between memory pictures cannot be located.

(5.) It may be noticed by some that throughout this article where the visual area has been mentioned it has been taken for granted that its situation is in the occipital lobe and cuneus. Among the fifty cases here collected are twenty-two in which the angular gyrus was destroyed without the production of any actual disturbance of vision. In two the angular gyrus and its subcortical tissue were together affected, with the production of hemianopsia—a symptom which may be justly ascribed to a lesion of the visual tract on its way to the cuneus; on the other hand, there are now over forty cases on record in which a lesion of the occipital lobe alone has produced homonymous hemanopsia; and the four cases collected by Seguin in 1886, to prove that the cuneus is the visual centre proper, have been added to by various authors, so that over twelve cases are now to be found. Chauffard and Bouveret have reported four cases of total blindness caused by lesion of both cunei at once, without lesions of the convexity. It seems, therefore, that an overwhelming amount of evidence can now be cited to prove that in man the visual area lies exclusively in the occipital lobe, and that the angular gyrus has no part in the function of vision. For pathology affords only very slender evidence of the existence of crossed amblyopia, and no cases have been reported since careful tests of the visual field have been made.

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[1] "Mental Evolution in Man," p. 36.

This conclusion, now accepted in Germany,[1] France,[2] and America,[3] has met with some opposition in England on the ground of the results of physiological experiment on apes. But it seems to be valid when tested by pathological evidence, and, as already stated at the beginning of this article, it is to this rather than to physiology that the final appeal must be made. Already the fact of the localisation of the visual area in the occipital lobe has been made the basis of several successful operations. It is therefore not unworthy of notice that these cases, collected for another purpose, confirm the conclusion that when an operation is undertaken upon the basis of a cortical visual disturbance alone the trephine should never be applied over the angular gyrus, but always posterior to it over the occipital convolutions.

[1] Nothnagel, 'Verhandl. des Cong. f. Inn. Med.,' Wiesbaden, 1887.

[2] Chauffard, 'Rev. de Medicine,' Jan., 1888.

[3] Mills, 'Trans. Cong. Amer. Phys. and Surgs.,' Washington, 1888.

Since cortical epilepsy beginning with hemiopic visual aura is a condition in which operative interference may be undertaken, this fact is not without importance.

It is also evident that areas concerned in the preservation of sensory memory pictures do not necessarily coincide with, but are usually more extensive than, the sensory areas proper. The cuneus is the sight centre, but visual memories are located in the occipital convolutions, and in the angular gyrus. We do not know the location of the auditory centre in man, but the auditory memories extend over the first and second temporal convolutions.

This is not to be regarded as extraordinary. It has an analogy in the motor sphere. For a lesion in the posterior part of the third frontal convolution obliterates the effort memories concerned in speech, producing motor aphasia without producing any actual paralysis in any muscle.

TABLE OF CASES OF SENSORY APHASIA,

NO. OF CASE.	AUTHOR.	REFERENCES	LESION. SITUATION.		POWER TO RECALL WORDS	
I	Bateman	On Aphasia, 1870, p. 73	T_1	P_2	Impaired	1
II.	Broadbent	Med.-Chir. Trans., 1872, p. 162	T_1	P_2	Impaired	2
III.	Wernicke	Aphas. Sympt. Comp., 1874 (10)	$T_1T_2T_3$		Impaired	3
IV.	Wernicke	l.c. (2)	T_1	P_2	..	4
V.	Lohmeyer	Arch. f. Klin Chir., xiii. 323	T_1	P_2 sm	Impaired	5
VI.	Troissier	Gaz. Med. de Paris, 1874, p. 25	$T_{1,2,3}$	A O_2O_3	.	6
VII.	Kussmaul	Ziemssen's Cyclop., xiv. p. 765	T_1T_2		Lost	7
VIII.	Kussmaul	l.c., p. 763		A $T_2—O_2$	Impaired	8
IX.	Gortz	Bullet. Soc. Anat., Paris, 1876, p. 81	T_1	P_2	Impaired	9
X.	Sabourin	Progrès Méd., 1877, p. 70	T_1T_2	P_2 sm	Impaired	10
XI.	Bulteau	Bullet. Soc. Anat., 1877, p. 262		P_2 A $O_{1,2,3}$	. .. .	11
XII	Broadbent	Lancet, 1878, i. 812	T_1T_2	P_2 A sm	.. ..	12
XIII.	Riedel	Dissert. Breslau, 1877	T_1T_2	A	. ..	13
XIV.	Fritsch	Wien. Med. Presse, 1880, p. 468	T_1T_2	P_2 A	. .. .	14
XV.	Ball & Seguin	Arch. of Med., 1881, p. 186	T_1	P_2 A sm	Impaired	15
XVI.	Chauffard	Rev. de Méd., 1881, p. 939	T_1T_2	P_2 A sm	. .	16
XVII.	Weiss	Wien. Med. Wochensch., 1882, p. 834	T_1	P_2 A sm O	. . .	17
XVIII.	Girandeau	Rev. de Méd., 1882, p. 446	T_1T_2		Good	18
XIX.	Claus	Irrenfreund, 1888, p. 82	T—O		Impaired	19
XX.	Claus	l.c., p. 88	T_1T_2		. . .	20
XXI	d'Heilly	Gaz. Méd. de Paris, 1888, p. 22	T	P_2 sm		21
XXII.	Webber	Boston Med. Surg. Jour., 1888, p. 580		P_2 sm	Impaired	22
XXIII.	Dejerine	Progrès Méd., 1880, p. 620		P_2 sm	Impaired	23
XXIV.	Schutz	Charité Annalen, xiii. 481	T_1T_2	A $O_{1,2,3}$	Impaired	24
XXV.	Balzer	Gaz. Med. de Paris, 1884, p. 97	T_1T_2	A O_3	Impaired	25
XXVI.	Rosenthal	Centralbl. fur Nerv., 1884, p. 1	T_1T_2	A sm	Impaired	26
XXVII.	Amidon	New York Med. Jour., 1885, p. 113	T_1T_2	P_2 A O_2	Impaired	27
XXVIII	Gunther	Zeit. f. Klin. Med., 1885, p. 16	T_1T_2	A $O_{2,3}$	. .	28
XXIX	Monakow	Arch. f. Psych., xvi. p. 160	T_1T_2	A $O_{2,3}$	. .	29
XXX	Seppilli	Functions local, p. 208	$T_1T_2T_3$		.	30
XXXI.	Seppilli	l.c., p. 205	T_1T_2	P_2	Impaired	31
XXXII.	Seppilli	l.c., p. 182	T_{1-3}	P_2 A O_{1-3}	. .. ..	32
XXXIII.	Petrazzani	Revista Sperimentale, xii. p. 285	T_{1-2}	Bilateral	. .. .	33
XXXIV.	Eichhorst	Corresp. Schw. Arzte, 1886, p. 606	T_1		.. .	34
XXXV.	Henschen	Neurol. Centralbl., 1886, p. 424 (2)		A	.. . .	35
XXXVI.	Henschen	l.c. (3)	T_1T_2	A	.	36
XXXVII.	Jastrowitz	Centrabl. f. Pract. Augenh., 1877, p. 254		O_{1-3}	Impaired	37
XXXVIII.	Perret	Clinique Médicale, p. 137	T_{1-3}	P_2 A O_{1-3}	.. . ..	38
XXXIX.	Hun	Amer. Jour. Med. Sci., 1887, p. 154		P_2 A	. .. .	39
XL.	Hitzig	Congress fur Inn. Med., 1887, p. 166	T_1T_2		.	40
XLI.	Sigaud	Progrès Med., 1887, p. 177		A	Good	41
XLII.	Reinhard	Arch. f. Psych., xviii. p. 244		O_{1-3}	Impaired	42
XLIII	Bernheim	Hecht. Thèse de Nancy, 1887		O_{1-3}	Impaired	43
XLIV	Wilbrand	Seelenblindheit, p. 180		O_{1-3}	Impaired	44
XLV.	Laquer	Neurol. Centralbl., 1888, p. 340	T_1	P_2 O_2	. ..	45
XLVI.	Macewen	Brit. Med. Journal., 1888, Aug. 11		A sm	Good	46
XLVII.	Freund	Arch. f. Psych., xx. 277	T_1T_2	A O_{1-3}	Impaired	47
XLVIII.	Wiglesworth	Liverpool Med.-Chir. Jour., 1887, p. 215	T_1	A sm	Lost	48
XLIX.	Franks	Med. Press and Circ., 1888, p. 20.	T_1T_2		Impaired	49
L.	Bullen	Brain, xi. p. 514	T_1T_2	A O_2	Lost	50

$T_{1,2,3}$ = First, second, and third Temporal Convolutions. $O_{1,2,3}$, First, second, and third Occipital Convolutions. P_2 = Inferior Parietal Convolutions lying between the supra marginal gyrus (SM.) and the angular gyrus (A.).

WITH LESIONS AND SYMPTOMS.

	POWER TO UNDERSTAND SPEECH.	POWER TO READ.	POWER TO TALK.	POWER TO REPEAT WORDS HEARD.	POWER TO WRITE AT WILL	POWER TO WRITE AT DICTATION	POWER TO COPY.	POWER TO READ ALOUD.	POWER TO RECOGNISE OBJECTS.	DISTURBANCE OF SIGHT
1	Lost	. . .	Good	. . .	Lost	. . .	. . .	. . .		None
2	Good	Lost	Good	. . .	Good	Good	. . .	Lost	Impaired	None
3	Impaired	Good	Fair	. . .	. . .	. . .	. . .	Impaired		
4	Lost	. . .	Good							
5	. . .	. . .	Impaired	. . .	Lost					
6	Lost	. . .	Impaired							
7	Lost	Impaired	Good	. . .	Letters, not words	. . .	. . .	Good, but did not understand		
8	Good	Impaired	Impaired	Impaired	Impaired	. . .	. . .	Lost		
9	Lost	.	Impaired	Lost						
10	Impaired	Lost	Very limited	Lost	Impaired					
11	Impaired	. . .	Impaired	Good						
12	Lost	Lost	Impaired	. . .	Lost					
13	Lost	Impaired	Impaired	. . .	Impaired					
14	Lost	Impaired	. . .	. . .	. . .	. . .	. . .	. . .	. . .	Right hemianopsia
15	Impaired	Impaired	Good	. . .	Impaired	Lost	Impaired	. . .	Impaired	None
16	Lost	.	Impaired							
17	Lost	Lost	Lost	Lost	Lost					
18	Lost	Good	Good	. . .	Good					
19	Impaired	. . .	Good	Good	Lost	Lost	Lost	. .	Impaired	
20	Lost	. . .	Good							
21	Lost	Lost	Impaired	Lost	Lost	Lost	Lost			
22	Good	Not tested	Impaired	. . .	Lost	Lost				
23	. . .	Lost, did not understand	Impaired	. . .	. . .	Good	. . .	Good, but did not understand		
24	Lost	. . .	Good	. . .	Impaired	Lost	Lost,			
25	Impaired	Lost	Impaired	Good	Lost	Lost	Lost			
26	Lost	Lost	Impaired	. . .	Name only					
27	Lost	Lost	Fair	. . .	Lost	Lost	. . .	Jargon	Impaired	
28	Lost	. . .	Impaired							
29	Lost	Lost	Good	. . .	. . .	. . .	.	. . .	Impaired	
30	Lost	. . .	Fair							
31	Lost	. . .	Impaired	Good						
32	Lost	. . .	Lost	. . .	. . .	. . .	. . .	. .	Impaired	Partially blind
33	Lost	. . .	Good							
34	Lost	. . .	Good							
35	Impaired	Lost								
36	Good	Lost	Impaired	. . .	Lost					
37	Good	Lost	Impaired	Impaired	Lost	. . .	.	. . .	. . .	Right hemianopsia
38	Lost	. . .	Impaired	. . .	. . .	. . .	. . .	. . .	. . .	None
39	Good	Lost	Impaired	. . .	Lost	Impaired	. . .	Lost	. . .	None
40	Lost		Good							
41	Good	Good	Good	Good	Lost	Much Impaired	Impaired	Good	Good	None
42	Good	Lost	Good	Good	Impaired	Good	Impaired	Lost	Impaired	Right hemianopsia
43	Good	Lost	Good	Lost	Lost	Lost	. . .	Lost	Impaired	Right hemianopsia
44	Good	Impaired	Good	. . .	Never learned	. . .	. . .	. . .	. . .	Right hemianopsia
45	Lost	Lost	Impaired	Lost	Never learned	. . .	. . .	. . .	Impaired	None
46	Good	Lost	Good	. . .	. . .	.	Lost	.	Impaired	None
47	Good	Lost	Good	Good	Lost	Lost,	Lost	Lost	Impaired	Right hemianopsia
48	Lost	Lost	Impaired	. . .	Lost	Lost	Lost	Lost	Impaired	None
49	. . .	. . .	Impaired	. . .	Impaired	. . .	. . .	. . .	. . .	None
50	Impaired	Lost	Impaired	Lost	Lost	Lost	Lost	Lost	Good	None

A CASE OF PERMANENT CONJUGATE DEVIATION OF THE EYES AND HEAD, THE RESULT OF A LESION LIMITED TO THE SIXTH NUCLEUS; WITH REMARKS ON ASSOCIATED LATERAL MOVEMENTS OF THE EYEBALLS, AND ROTATION OF THE HEAD AND NECK.

BY A. HUGHES BENNETT, M.D., AND THOMAS SAVILL, M.D.

THE following case is one of great interest, and of extreme rarity. The patient during life suffered from *permanent* conjugate deviation of the eyeballs and head. This was diagnosed before death to be the result of a lesion of the sixth nucleus on one side. On post-mortem examination a minute softening was found occupying, and limited to, that centre.

Elizabeth G., aged sixty-seven, a domestic servant, was admitted into the Paddington Infirmary on October 11th, 1887. Her family history was unimportant. The patient had always enjoyed good health till the present illness. In August, 1887, having gone to bed one evening in her usual condition, she awoke next morning to find that she was afflicted with complete paralysis of the left upper extremity, otherwise she was quite well. This condition remained unchanged for two months, when one morning on waking, she found in addition, that both her eyes were turned towards the right side, so that she could see nothing in front of her, and that her head was fixedly rotated towards the right side. Three days afterwards she came into the Infirmary. On October 13th her condition was briefly as follows. The patient was weak and confined to bed. Her intelligence appeared normal, and she replied to all questions with accuracy. There was very trifling paresis of the left side of the face. The left upper extremity was motionless throughout, from the shoulder downwards. There was no muscular wasting, and the sensibility of the skin was everywhere intact. The reaction of the tendons and muscles to percussion was comparatively increased in the left arm. Both eyeballs were firmly and permanently fixed towards the right side, and the strongest efforts of the will could barely bring them towards the middle line, and in this the left eye was specially deficient. When each eye was tested separately the right could be moved to, and even a little beyond, the middle line, but the left did not reach that point. Both eyeballs converged when an object was brought close to them. The pupils were equal and normal. The head was firmly and permanently rotated towards the right, and could not voluntarily be brought into a straight position. The chin was tilted forwards and upwards, due to contraction of the left sterno-mastoid muscle. Both lower extremities, although weak, were equal and apparently not paralysed. The knee-jerk on the left side was somewhat more lively than on the other, but was not excessively increased. The special senses were practically normal, although both hearing (especially in the left ear) and vision appeared to be somewhat impaired. Ophthalmoscopic examination showed the media to be slightly opaque, and the fundi practically healthy. There was no hemiopia. The general functions and organs of the body were normal. From this date till the death of the patient, about a month later (November 14th), the condition remained unchanged. She gradually became weaker, and died comatose, exhibiting all the symptoms above described.

Post-mortem Examination.—With the exception of the nervous system the organs of the body were essentially healthy. *Nervous System.*—The brain weighed forty-six ounces. The dura was thickened and slightly adherent to the cranium. There was a large quantity of sub-arachnoid effusion, and over the surface of the brain a considerable amount of venous congestion. The sinuses were normal. The arteries at the base were thickened and atheromatous. The convolutions of the brain were of normal appearance except the right ascending frontal. This throughout its whole extent, to within about an inch of the longitudinal fissure was pale flattened, and quite soft to the touch. At the upper part of this convolution, and close to the marginal gyrus was a recent superficial meningeal hæmorrhage about the size of a sixpence. On making a transverse vertical section through the ascending frontal convolution, the softening was seen to be of triangular shape, the base occupying the area above described on the surface, and the apex touching the summit of the internal capsule. This was about three-quarters of an inch in thickness. A secondary descending process could be followed downwards through the right internal capsule and crus cerebri. The brain was otherwise healthy. On removing the pons and medulla, and making a transverse section exactly at their junction, a small flat circular patch of softening, about the size of a large mustard seed, or more accurately about one-tenth of an inch in diameter, was seen occupying the position of the left sixth nucleus, and limited to it without apparently involving the neighbouring structures, such as the facial fibres (Fig. 1). Otherwise, to the naked eye the

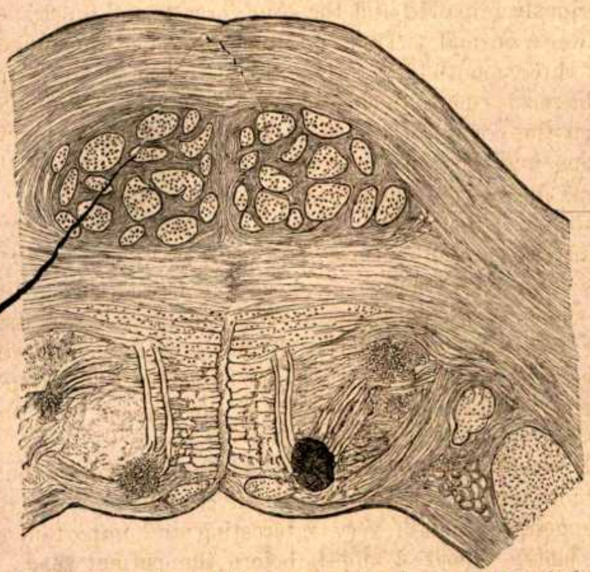

FIG. 1.—Transverse section of the pons at the level of the sixth nucleus showing a limited softening of that centre.

appearances of the pons and medulla were normal. These, with the cord and other nervous structures, were reserved for subsequent and more complete examination, but unfortunately owing to a mishap they were destroyed. The above-described softening in the pons was, however, carefully observed at the post-mortem examination, and an accurate drawing made of it at the time, which is here reproduced. The only structures which were not lost were the two sixth nerves. These were examined with the greatest care, and several independent observers—including Drs. Hebb (who kindly made the sections) and Byrom Bramwell—agreed as to the microscopical appearances they exhibited. The right nerve was perfectly healthy. The left here and there showed very slight traces of degenerative atrophy.

Commentary.—The first phase of this patient's condition was paralysis of the left upper extremity. Having been previously in good health, she suddenly during the night lost all power in the entire arm from the shoulder downwards. The muscles were all equally affected; there was no wasting or loss of sensibility, and some weeks afterwards the tendon reflexes were exaggerated. This was associated with very slight paresis of the left side of the face, but the left leg was not perceptibly affected. At no time were any convulsions observed; the intelligence was not seriously impaired and the other organs and functions of the body were normal. This condition remained unchanged until death, three months after the original onset of the symptoms. The diagnosis made during life was that the pyramidal tract between the cortex of the descending frontal and parietal convolutions, and the upper part of the internal capsule on the right side, was interrupted, slightly encroaching on the facial area. The post-mortem examination showed this surmise to have been substantially correct, and not only were the fibres of the corona radiata underlying these convolutions softened, but almost the entire cortex of the ascending frontal convolution was itself in a similar condition, So far the clinical symptoms perfectly harmonised with what modern experience would lead us to expect from the anatomical distribution of the disease, and therefore call for no further remarks.

The second phase of this case is equally definite, but being much more uncommon, merits careful consideration, as it serves to demonstrate certain very interesting and important physiological facts. About a month before the patient died, having been in her usual condition, she awoke one morning to find that both her eyeballs, as well as her head and neck, were rigidly directed towards the right side, and that no voluntary effort on her part permitted her to bring them, especially the left eye, as far as the middle line. There was marked stiffness and contraction of the left sterno-mastoid muscle. The two eyes in conjunction could not be made to move towards the left, but the right eye was capable of performing a lateral movement towards the left in the act of convergence with both eyes fixed on a near object, or when tested by itself with the left eye closed. In short, there was complete conjugate paralysis of the eyeballs on the left side, the deviation of these towards the right being due to the unopposed action of the healthy muscles on that side, while convergent action of both eyes remained intact. The left external rectus muscle was completely and permanently paralysed for all purposes. The internal rectus was only affected during the act of conjugation, and contracted normally under all other circumstances. With the exception of the conditions described the patient was otherwise well, and *these symptoms in no way improved, but remained permanently unchanged till her death a month afterwards.*

It is interesting to note that the diagnosis which was hazarded during life was afterwards proved by post-mortem examination to have been correct, namely, that the ocular phenomena were due to a lesion of the left sixth nucleus. It was clear that the conjugate deviation was not the result of fresh extension of the disease in the right cerebral hemisphere, otherwise it would have been of a temporary character and not so complete and permanent as it was in this instance. Any doubt which may have existed during life as to whether the deformity was of a spasmodic or paralytic nature was definitely settled by the post-mortem examination in favour of the latter. The symptoms in no way improving suggested either interruption of the commissural fibres between the third and sixth nuclei, combined with a second lesion in the motor tract above the latter, or disease of the sixth nucleus itself, the second hypothesis being much the more probable of the two. That a lesion should be so small and so strictly limited to so minute a centre as to annihilate its functions without complicating the neighbouring structures, and notably the facial fibres, is as remarkable as it must be rare, and the result will constitute an important fact in favour of those who, like Duval, Laborde, Graux and Landouzy, have maintained that the sixth nucleus is the reflex centre presiding over that complicated automatism by which the eyeballs, head and neck move harmoniously in concert.

By conjugate movements of the eyeballs is understood that action by which, under an appropriate stimulus, the two eyes move together in turning towards the right or left. This may be a purely reflex or automatic act, or it may be the result of a voluntary impulse, the former being carried on through the agency of a complicated mechanism in the pons, the latter, of course, originating in certain portions of the cortex cerebri. When both eyeballs look in the same direction, it is due to the contraction of appropriate muscles, and as the external and internal recti are anatomically supplied by different nerves, it is obvious that for conjugate action there must be some association between the divergent nervous supply, in order to account for the concerted action. The details of the mechanism by which this is effected may be shortly summarised. The internal recti muscles are supplied by the third, and the external recti by the sixth pair of nerves, each originating from their respective nuclei. When the two eyes look in one direction there is, of course, contraction of the external rectus of one eye, and the internal rectus of the other, which is effected by the action of their corresponding nuclei, namely, the sixth on one side and the third on the other. The simultaneous functionating of these two anatomically distinct centres shows that there is a physiological connection between the two.

The manner in which this is effected is as follows: a sensory stimulus, such as a sudden flash of light, or a sound on one side, will reflexly cause both eyes to turn in that direction. This sensory impression is received first by the eye or ear nearest the light or sound, so that through the optic or acoustic nerves on that side it is conveyed to the corresponding sixth nucleus, exciting there a motor impulse which is directly carried by the sixth nerve to the external rectus, thus causing the contraction of that muscle. This eye takes the lead in the action and moves outwards. Almost simultaneously the motor impulse is directed by a crossed communicating track to the third nucleus of the opposite side, and through this by fibres in the third nerve to the corresponding internal rectus muscle, which also contracts. Thus both eyeballs are directed towards the light or sound, the sixth nucleus being the reflex centre by which the combined act is carried on. The afferent impulse is derived from the optic or auditory nerves, and the efferent tracts are, first, the sixth nerve on the same side, and second, the crossed fibres joining the sixth nucleus to the third nucleus of the opposite side, as well as the same fibres continued into the third nerve.

The accompanying diagram (Fig. 2) attempts schematically to show the mechanism by which this process is

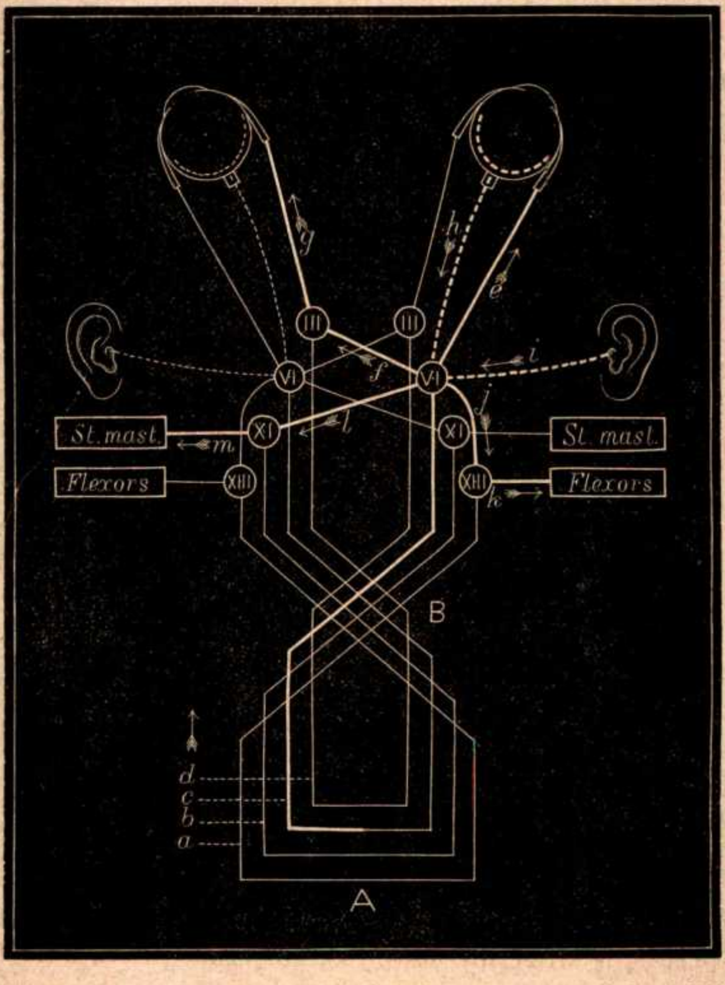

FIG. 2.—*Diagram showing mechanism of conjugate movements of the eyeballs and rotation of the head and neck.*

The dark lines represent the voluntary motor tract from the cortex to the sixth nucleus, as well as the other efferent tracts from this centre by which conjugate movements of the eyeballs, head and neck are accomplished. The dotted lines indicate the afferent tracts from the eye and ear to the sixth nucleus. The arrows show the direction of the different nerve currents.

A. Cortex Cerebri; ***a.*** Voluntary tract from cortex to cervical ganglia; ***b.***

Ditto to eleventh nucleus; *c.* Ditto to sixth nucleus; *d.* Ditto to third nucleus; B. Decussation of voluntary motor tracts in medulla and pons; III. Third nucleus; VI. Sixth nucleus; XI. Eleventh nucleus; XIII. Upper cervical ganglia; *e.* Motor fibres from sixth nucleus to external rectus; *f.* Crossed tract between third and sixth nuclei; *g.* Motor fibres from third nucleus to internal rectus; *h.* Afferent tract from retina to sixth nucleus; *i.* Afferent tract from ear to sixth nucleus; *j.* Communicating tract between sixth nucleus and cervical ganglia on the same side; *k.* Motor nerve from cervical ganglia to flexors of neck; *l.* Crossed tract between sixth and eleventh nuclei of opposite sides; *m.* Motor nerve from eleventh nucleus to sterno-mastoid muscle.

effected. The afferent auditory tract (*i*) reaches the sixth nucleus (VI.). From thence extend the efferent tracts (*e*) to the external rectus of the same eye, and (*f*, *g*) by the third nucleus (III.) to the internal rectus of the other. The arrangement has been compared to the reins used in driving a pair of horses. When one (*i* or *c*) is pulled, owing to its bifurcation (*e* and *f g* at VI.) both the animals' heads (the eyeballs?) are drawn in one direction. The afferent tract from the retina (*h*) is also associated with the sixth nucleus, and, when appropriately stimulated, produces similar reflex conjugate movements. Some physiologists believe that in addition to the mechanism just described, there is another special centre which intervenes between the retinal and auditory nuclei and the sixth nucleus, and this they place in the superior olivary body. It does not appear that such an assumption is necessary, as the more simple arrangement answers all physiological requirements.

That some such disposition as the foregoing must exist is proved by anatomical, physiological and clinical experience. Graux[1] has in the cat actually demonstrated connecting fibres between the third and sixth nuclei. By experiments on dogs the same observer has shown that when the six nucleus is artificially destroyed, conjugate paralysis of eyeballs on the same side, with deviation in an opp direction, is the result. Finally, clinical facts, and the case under consideration, indicate that the s clusion can be drawn from disease of this gangl

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[1] Graux (Gaston), De la Paralysie du Moteur oc Deviation conjugée. Paris, 1878.

Thus the mechanism of reflex conjugate movements of the eyeballs may be said to be organised in the pons, and the special centre through which it is carried on is the ganglion to which the name of sixth nucleus has been given by anatomists. Irritation of that group of cells causes conjugate spasm towards the same side, and its destruction produces conjugate paralysis in the same situation, with deviation of the eyeballs in the other direction, the result of the unopposed action of the healthy muscles.

An interesting feature connected with conjugate action is exemplified by this case, namely the fact that although the muscles connected with this associated phenomenon may be paralysed, it does not follow that they lose their function for other purposes. Here for example the internal rectus of one eye was inactive to either the voluntary or reflex impulse of conjugate movement, yet it acted normally when the other eye was closed, and to convergent requirements. In short, this muscle was only cut off from the motor impulses derived from the sixth nucleus, while those connecting it with the third nucleus remained intact (Fig. 2). Therefore all the movements of the right eye were normal except during the act of conjugation, when the internal rectus was immovable. The external rectus of the left eye, on the other hand, was completely and permanently paralysed for all purposes, whether voluntary or reflex, for reasons which the preceding argument must have made obvious.

Although conjugate movements may in this manner be regarded as of purely reflex production, the same physiological condition may be accomplished as a voluntary act. Impulses from the cortex in connection with the pontine entres are capable of causing the eyeballs to move to the ht or left at will. The posterior portion of the frontal olutions are believed by Ferrier to be the area in which originate, and both experiment and clinical observation indicate this as the most probable region. Grasset believe the angular gyrus and its neighbourhood at of this process. Whatever the exact area, can be little doubt that conjugate movements have a definite localisation in the cerebral cortex, as is abundantly shown by experimental and clinical observation. This cortical centre is connected with the nuclei in the pons by fibres which decussate immediately above them, and the point at which this crossing takes place is believed to be at the corpora quadrigemina. By this means voluntary impulse can be transmitted, and the eyes and head moved conjugately in any direction that may be desired (Fig. 2, *c*). Irritation of the cortex of the brain artificially or by disease causes conjugate spasm. This has been produced in animals, and is frequently seen as one of the initial symptoms of an epileptic seizure. Destructive lesions, again, cause conjugate paralysis, which has also been produced experimentally, and is frequently met with clinically, usually in association with severe cases of hemiplegia.

The foregoing phenomena, whether reflex or voluntary, are further rendered more complex by the circumstance, that on the application of an appropriate stimulus, in addition to the eyes, the head and neck also may act conjugately and be directed towards it, the former being so rotated and inclined as to look over the shoulder.

This conjugate movement of the head with the eyeballs is carried on after the same manner and through the same centres as have just been enumerated, only there is superadded a still more complex series of connections. To the mechanism already formulated, there must be further associations with the sixth nucleus, as the primary starting-point of the special function. Commissural fibres must connect it with the centres for both the rotators and flexors of the head and neck. Movement of the head to one side is effected partly by rotation, and partly by inclination or flexion. The muscles which chiefly come into play for the first purpose, are the sterno-mastoid and trapezius of the opposite side, which are supplied by the spinal accessory nerve, and for the second the superior oblique, splenius, recti and possibly other muscles on the same side, supplied by cervical nerves, the combined action of which rotates and inclines the head over the shoulder. The diagram (Fig. 2) attempts to show how these various connections may take place, and the relations that may exist between the sixth and eleventh nuclei of opposite sides, and between the former and the cervical ganglia on the same side. An impulse, whether voluntary (*c*), or from sensory impressions, (*h* and *i*) acting on the sixth nucleus (VI.), through its influence will cause contraction of the external rectus (*e*) and the flexors of the neck (*k*) through connections with the cervical ganglia (XIII.) on the same side; and simultaneously contraction of the internal rectus (*g*), and the sterno-mastoid (*m*) through the eleventh nucleus (XI.) of the opposite side. The result of this is a conjugate movement of both eyeballs, and a rotation and inclination of the head towards the side of the sixth nucleus which receives and distributes the impulse. Destruction of this centre would of course have exactly the opposite effects.

Such then is an attempt to explain the mechanism of this highly complex physiological phenomenon. Conjugate movement of the eyeballs and head, an act which partly by inheritance and partly by education, has become organised in the nervous centres, reflexly in the pons, by volition in the cerebral cortex. The two are intimately connected by connecting elements which decussate in the pontine region, possibly at the corpora quadrigemina. The apparently simple act of head and eyes looking in concert towards an object, is carried on by a diverse system of nerves and muscles which have no anatomical relation one with the other, but all of which, excited by a suitable stimulus, and through the agency of a common centre, act harmoniously together to effect the physiological purpose desired. Any disturbance of this centre involves derangement of the conjugate phenomenon as a whole, but leaves the individual elements, with exception of the sixth nerve, to act normally for any other purpose, and in any other form except that of conjugation.

Any portion of the entire conjugate tract from cortex to muscle may be interrupted by disease, and give rise to corresponding symptoms. These, if properly interpreted, enable us, during the life of the patient, to diagnose with considerable accuracy the nature and locality of the lesion. The clinical facts may be briefly summed up as follows:

1. An irritative lesion at A, the cortex cerebri (Fig. 2), at *c*, the area of conjugate movements, on one side, causes conjugate spasm of the eyeballs and head towards the opposite side from the lesion, with deviation away from the diseased side of the brain. This is seen in experimental stimulation, and in some cases of epilepsy. Destruction of the same region induces paralysis in the same distribution, with deviation of the eyeballs in a contrary direction, namely towards the diseased hemisphere, owing to the antagonistic action of the healthy muscles. This also may be produced experimentally and is met with in severe cases of hæmorrhage or softening accompanying the early stages of hemiplegia. It is in such cases usually a temporary symptom, as the various nervous connections soon enable the other side, by opening out new channels, to perform in this respect the functions of the damaged hemisphere. The same results follow interruption at any portion of the motor tract between A and B, that is, between the cortex and the decussation of fibres in the pons. In such cases also the paralysis of the eyeballs is rarely complete. They may appear straight at rest, but there is a difficulty in moving them beyond the middle line. The internal rectus however converges readily in association with the other eye, and also by itself when isolated. The affected external rectus can also move the eyeball outwards when the other eye is closed, as the corresponding nucleus and its voluntary connections are unimpaired. Usually there is no evidence of paresis of the muscles of the neck, and little apparent deviation of the head, for obvious reasons.

2. An irritating lesion, occurring anywhere between B and VI., that is, between the decussation of the motor fibres in the pons, and the sixth nucleus, causes exactly similar symptoms, as No. 1, only the direction of the deviation is reversed, in other words it is towards the side of the lesion. In destructive disease of the same region, the paralysis of the muscles is on the same side as the lesion, and the deviation away from it. The eyeballs can be voluntarily brought to the middle line but no further, and the internal rectus can be made to converge in association with the other

eye, and by itself when alone. The external rectus moves the eyeball outwards when the other eye is closed. The symptoms as in No. 1, are temporary in character, lasting at most for a few days, and the deviation of the head is little if at all observed.

3. Irritation at VI. or at the sixth nucleus itself, causes conjugate deviation of the eyeballs, with rotation and flexion of the head and neck towards the diseased side. Destruction of the centre causes paralysis of the same muscles, with deviation of the eyeballs away from the side of the lesion. In this case the conjugate deformity of the eyeballs is permanent, especially of the eye on the same side as the diseased nucleus, for the nervous connections with the centre which accomplish the conjoint action are broken, and not readily replaced. For the contrary reason the rotation and flexion of the head and neck is only temporary, if seen at all. There is total palsy of the external rectus of the eye nearest the lesion, the eyeball deviates inwards and cannot be moved outwards as far as the middle line, even when the other eye is closed. The other eye may be brought as far as the middle line, but not beyond it in conjugate association. If, however, both eyes being open, it is brought to converge, or when tested by itself, the other eye being closed, the internal rectus will be found to contract inwards beyond the middle line. This is owing to the fibres which supply the muscle from the sixth nucleus of the opposite side being cut off, and those derived from the third nucleus on the same side being intact. It is obvious that, if a lesion existed in the commissural fibres between the third and the sixth nuclei, accompanied by a second in the motor tract above the latter, that similar results would be produced.

4. A destructive limited lesion immediately in front of the sixth nucleus, involving the fibres of the sixth nerve, causes paralysis of the external rectus of the corresponding eye only, the function of the internal rectus of the other eye remaining intact. There are the usual signs of paralysis of the trunk of the sixth nerve, namely, internal strabismus of the affected eye, and secondary deviation of the other.

The case under notice is an example of No. 3, and this, owing to the preceding considerations, was diagnosed during the life of the patient. That the deviation of the eyeballs was towards the right, and that the lesion was found in the left sixth nucleus, leaves no doubt that the affection of the ocular nerves was a paralytic one. The association between disease of the centre and the conjugate affection is also proved, and if further evidence of lesion of this ganglion is required, it is to be found in the commencing secondary degeneration of the sixth nerve on the affected side. The main difficulty in this case was to account for the *permanent* rotation of the head towards the right side. This could not be owing either to the lesion of the right cortex, or to the destruction of the left sixth nucleus, as in either case such a condition would have been temporary, even if it ever existed at all from such causes. If even under these circumstances weakness of the sterno-mastoid and trapezius and the left flexors of the neck had been produced, the result would have been most fleeting, and soon compensated for by the functionating of the other channels. The most plausible explanation seems to be, that the deformity was caused by active spasm of the left sterno-mastoid muscle, due to direct irritation by the lesion in the sixth nucleus, upon the neighbouring eleventh nucleus, thus causing contraction of the muscle on the same side, through the spinal accessory nerve. Hence the head was tilted over the right shoulder. In favour of this view is the fact that the sterno-mastoid muscle during life was so rigidly contracted that the head could not be brought into the middle line either by voluntary or passive effort, and this condition remained permanent till death.

The facial has very close relations to the sixth nucleus, the fibres of the former coursing round the base of the latter. In this case there was slight paresis of the left side of the face, but this was due to the cortical lesion, and existed prior to the date of the nuclear disease, and moreover the softening was seen to have left the facial fibres intact. There was no evidence of facial rigidity or spasm, which might have occurred from irritation of the seventh nucleus, but this, even if it existed to a slight extent, may not have been observed, and may have been masked by the other condition of cortical paresis.

The chief subject of regret in this case was the unfortunate loss of the morbid specimens. Happily the nature and exact relations of the lesion were sufficiently observed and figured at the time of the post-mortem examination, as to have served for the foregoing purposes. But opportunities for more delicate investigations, such as the histological characters, and a research into the possible secondary degenerations, for which the parts had been specially reserved, were lost.

AORTIC ANEURYSM AND INSANITY.

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WITH these aneurysms may be no marked modification of mental state, or the case may be too complicated for minute analysis.

In some cases of thoracic aneurysm, there is either the production of mental disorder, or distinct modification of the pre-existent mental state. In the one case, this arises in persons predisposed to mental disorders; in the other, in those already insane; and in each as an indirect result of the effects of the aneurysm. These effects are chiefly due to one or more of several factors, varying according to the position, size and relations of the aneurysm, and the preceding mental and physical state of the patient in different cases.

One factor consists of the morbid afferent impressions coming from the altered vessel itself, and from the (usually) pulsatile, expansile tumour it now forms.

Another factor is the circulatory disorder, induced by thoracic aneurysm—both the general disorder of circulation, and that of the head more particularly. When the sac remains patent and not occupied by firm clot or laminæ, not merely is there the diversion and tumultuous rush into the sac of a large share of the blood-stream—thus interfering with the calm and equable supply to brain and other parts—but also the aneurysm in many cases presses upon blood-vessels: if it presses on an artery, checking or stopping more or less, the arterial supply to some parts (*e.g.*, the brain), and as a correlative result, making that to other parts correspondingly fuller and more active; if it presses on a vein, damming back the blood-stream in the latter, and tending to produce passive congestion in the region it drains, as well as coagulation in the vein itself. And to this may be added compression of lymphatic vessels and its results. This leads us to, indeed is a manifestation of, another and third factor, namely, the compression of important structures by the aneurysmal tumour. These structures we may broadly divide into three groups: one consisting of the blood and lymphatic vessels already mentioned; another being the nerves and nerve-ganglia in the thorax, relating specially to the vegetative or visceral life—chiefly the vagus and its branches, the phrenic and the sympathetic. The important symptoms whether irritative or inhibitory which may thus arise—be they vaso-motor, cardiac, pulmonary, respiratory, gastric or hepatic—only require bare mention. The third group comprises structures such as the thoracic bones and muscles, and nerve fibres to and from the trunk or limbs. The painful, or spasmodic, or paralytic conditions arising from compression of these may be the source of morbid material for mentation, and assist in the production or modification of mental disorder.

When with aneurysms of the thoracic aorta, mental symptoms appear to arise indirectly as already stated, they are usually of the following general characters: extraordinary illusions, hallucinations and delusions of persecutory and hypochondriacal nature. They may be delusions as to being annoyed and tormented, or as to bodily injuries, which may vary extremely in particulars, and often are most minutely described and attributed each to some very special and peculiarly distinct action or operation on this or that part of the frame of the patient, as, for example, by animals which have gained access thereto; or delusions as to hostile, occult influences working against the patient, culminating in assertions as to the particular effects of galvanic batteries, mesmerism, and so on—associated, perhaps, with vivid hallucinations, especially of hearing. The above persecutions and injuries are usually attributed to influences originating from outside, *e.g.*, to invasion by animals, or more often to the working of persons hostile to the patient, whom he has formerly known, and who have followed him to where he is now and is at their mercy—persons against whom he demands police or magisterial protection, and that on them condign punishment shall fall; although delusions of ill-treatment by those around, and in charge of, him may co-exist, or replace the former, and may lead to acts of violence, or even homicidal attempts. Though present in some cases, systemisation of the delusions may be absent, or feeble, or incomplete.

The emotional state is usually a mixture of depression and irritability, but as a rule there is not melancholia, properly speaking. There may be a gloomy, sombre, taciturn, or a sullen, morose state; or one of anguish, distress, anxiety, and free complaint as to annoyances, damages, persecutions, with desire to seek redress, and expostulation against detention within reach of the foe.

With this subject I dealt briefly in my Gulstonian Lectures[1] at the Royal College of Physicians, London, and gave a concise clinical summary of several of the cases which follow. And with regard to

Abdominal Aortic Aneurysms, I also stated[2] that "in three insane patients under my care, the cause of death was aneurysm of the abdominal aorta. It is perhaps unnecessary to give summaries of these cases, especially in view of the limitations imposed by waning space. Suffice it to say that in these cases of abdominal aortic aneurysm we find illustrations of the interpretation put by the insane mind upon the symptoms, especially the pains or discomfort arising from the pressure and other effects of the aneurysm; and we find that interpretation taking the form of delusions of definite types, or showing a tendency thereto. We trace in these abdominal aneurysmal cases at least the same character of delusions as to local bodily injuries, damages, personal injuries, hostile influences and effects, and delusions of mingled persecutory and hypochondriacal types, as in some thoracic aneurysmal cases. Yet the abdominal aneurysms were complicated with more or less heart disease, and therefore, were not pure cases;" moreover, the thoracic aorta, also, was somewhat affected in them.

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[1] 'On Insanity in Relation to Cardiac and Aortic Disease and Phthisis,' 1888: H. K. Lewis.

[2] *Ibid*, p. 64.

In the following pages eight cases of large thoracic aortic aneurysm are mentioned, and summaries of the cases suitable for the present purpose are given; as also are those of three cases of fatal abdominal aortic aneurysm; or eleven cases in all.

THORACIC AORTIC ANEURYSMS.

CASE I.—J. G. Six and a-half years after admission and at the age of thirty-six, a soldier under my care died, whose insanity was stated to have come on nine months before admission and, attributed to the effects of tropical East Indian climate and to intemperance, it followed his reception into a military hospital for the effects of heavy drinking. Shortly before he came here were mental excitement, rapid flow of thought, voluble utterance, disjointed and erroneous statements, delusions such as that he was hunted by his regimental comrades for horrible purposes, and under the influence of which he attempted suicide.

On admission.—Bubo scars in both groins; a few old, reddish, other scars. Depressed, solitary and taciturn, he evinced the same, and above-mentioned, delusions, and in conversation was somewhat incoherent, quarrelsome and reviling. He passed through attacks of catarrh and of jaundice. Later on, more cheerful and less quarrelsome, he did a little work, and is recorded to have suffered from "rheumatism;" but still later, was more apathetic and disinclined for work or amusement.

Cardiac hypertrophy (left ventricle) was found; and disease of both mitral and aortic valves (*bruits*, &c.), and aneurysm of thoracic aorta with double bellows sound. There had previously been a systolic *bruit*, propagated over the aortic arch and along the subclavian arteries.

The aneurysm increased; on the chest surface was a pulsatile expansile swelling. At each beat the thoracic parietes were visibly raised over an area extending from the first to the fourth rib on the right side, and from the right sternal edge half way to the shoulder; in this area was percussion-dulness, gradually shading off from a line opposite to the right third costal cartilage. Dyspnœa increased to orthopnœa during the last part of life.

Sweating in bed occurred even during cold weather. Œdema affected the eyelids, and brawny œdema the face; and, notwithstanding his favourite dextral semi-prone decubitus, the left eyelid was the more œdematous of the two. It was from the left nostril only that epistaxis occurred on one or two days towards the close. The two radial pulses were equal; the temperatures the same in the two axillæ; the pupils equal, or at times the left slightly the larger; both being irregular in contour and widest in their transverse diameter, the right one oblong, the left oval, in shape. Though not extremely affected, the voice was hoarse and at times slightly croupy; so was the cough. The full and fairly compressible pulse was at times slightly jerking. The subcutaneous veins were enlarged opposite the diaphragm. Latterly, were turgid lividity of the face, coldness and livor of hands. Œdema and serous effusion were limited to the pleural cavities, face and eyelids (pericardium adherent). Only late and rare dysphagia; no "corded" feeling anywhere.

During, and for a time prior to the attainment of large size by the still growing aneurysmal tumour, were peculiar delusions of being tormented, grievously hurt—"destroyed"—and his bones bored into, by snakes, scorpions, weasels, &c., in trunk and limbs. Once he had "seen" and once he had "heard" them. To quote from notes: Says his body is full of snakes, scorpions, rats and weasels, which eat him up and gnaw on the bones. "They are the most horrible 'varmint' ever known." Later: often feels his hand and head "falling off," has "pains all over," worse in trunk and head, equally on the two sides, and boring pains through both jaws. "Is eaten up alive; some of the 'varmint' make sores which others eat every day." "It began the same way six or seven years ago." "Millions of snakes and scorpions got inside and began eating his body. They now push out his ribs, first at one side, then at the other, and eat them, and finally level them down. They also push out and eat the jaws."

Later on, being short of breath, he says it is forced out by the "varmint" inside him; his "chest is all sores, and every inch of his body is the same." About this time were profuse clammy sweat and interscapular tubular respiration. Later, respiration was sudden, rapid, audible, as of one taking breath after prolonged strain or exertion. Still later, respiration tracheal, wheezy; expiration long drawn out.

Subsequently, says the top of his head, especially its left side and his ribs "are split out." Veins of neck distended, ears turgid, face turgid and sweating, hæmorrhage from left nostril, brassy hoarse cough, tracheal respiration; and once complained of dysphagia.

Once subsequently, he says the vermin ("varmint") "carry pocket knives, and during the last twenty-four hours have been scraping his ribs, first on the edge and then on the broad, and finally they bend them and go through the operation of breaking them." Giddiness, "reeliness," and "a blindness" (as he called them), dyspnœa, orthopnœa, cold and purple hands, ear-tips, nose, and lips, preceded somewhat sudden death.

Abstract of Necropsy.—The intact aneursymal sac contained an enormous amount of clot and blood (fl℥30 were collected, and much more escaped). Saccular, and affecting the right side of aortic arch, the aneurysm occupied much of the front of the right side of the thorax, and had eroded ribs, cartilages, sternum, muscles, and mediastinal tissues, more or less over the inner portions of first three right intercostal spaces and adjoining parts. In size it was about five inches wide, four in depth, and four from before backwards; it did not press on the spinal column, but *did* on the right lung, right pulmonary vein, and (perhaps) right bronchus, on right vagus nerve, superior vena cava, and right auricle of heart; only slightly on the pulmonary artery, and not (at least in the quiescent state) on the œsophagus or trachea. Rugous, the aneurysmal walls of the aorta were atheromatous, presenting yellow fatty points, &c. The sac began immediately above the enlarged sinuses of Valsalva, expanded to the right, to the front, and backwards, but especially to the right, the left third of the circumference of the aortic wall not being involved in the aneurysmal expansion. The edges of the mouth of the aneurysm were irregularly undulate; the orifice was 2 × 2½ inches in diameter.

The greatly hypertrophied left chambers, chiefly the ventricle, with rounded broad apex of the heart, were in contrast with the but little altered state of the right ones. The aortic and mitral valves were diseased, but fairly competent. The pericardium was adherent, the heart was heavy.

Spleen, 7oz., firm, capsule adherent and irregularly thickened, a firm cartilaginoid patch on it. Liver, 52oz., adhesions to its capsule, somewhat "nutmeggy." Kidney, 4¾ and 5oz., very slightly granular. Lungs, right pleural cavity, fl. oz. 25 serous fluid, with some lymph; left pleural cavity, fl. oz. 17 serous fluid. Lungs partially carnified. Skin of body sallow, parchment-like.

CASE II.—J. K. Six months after the onset of his insanity, a soldier, who had served long in India, came under my care for three months, until his death at the age of thirty-four. Admitted with disease of the heart and of the aortic valve, chiefly obstructive, but at times indicated by a double murmur, he also suffered from thoracic aortic aneurysm of some standing. As the chief facts from the necropsy will be given there is less need to state the various and varying physical signs observed at different parts of the course of the case.

The certificates under which he was admitted stated that he had the delusions that he was plotted against, conspired against by some women and a Captain S——, and also by the men of his regiment; that in an excited way he demanded immediate trial or redress, refused to answer questions about himself, and was restless and excited in appearance.

On Admission.—The delusions continued, but he was taciturn and complained of pain in chest and dyspnœa (much relieved by treatment), with congestion of lung, and expectoration, at first clear, then streaked with blood. In the groin, a bubo scar. So the case went on. He said the men watched and talked about him in India, and that therefore he was sent into hospital. Besides pulmonary congestion and blood-streaked sputa, were now constant hacking cough and occasional severe pain in scrobiculus cordis, right infra-clavicular and sub-scapular regions. Later on, were orthopnœa, anasarca of lower limbs—relieved by puncturing legs and free purgation—icteroid conjuctivæ, sallow hue of skin, moaning restlessness, malaise, bronchial and pulmonary congestion—at first more on left, then more on right side; aneurysmal bulging at second and third right ribs and spaces, pain chiefly in chest and right shoulder. He retrograded steadily, suffered much from orthopnœa and restlessness, declared that a galvanic battery was constantly applied to him by Captain S—— and others; this they "took off" (he said) on the approach of the asylum medical officers. He asserted that he distinctly heard them talking about him and what they would do to him, and saying "give it him," &c., and often he urgently pressed to have the police brought in or to be allowed to make oath before a magistrate as to these hallucinations, and secure the protection to be afforded by the law against his persecutors. Wearing a pained and anxious expression, gloomy, dejected, anguished, he was an apt example of the mental and physical suffering engendered by aortic aneurysm.

Œdema of legs set in a week before death and extended to the abdomen and scrotum. Vomiting, anorexia, icteroid and livid countenance (incisions in legs, hydragogue cathartics, stimulants). Painful delirium and orthopnœa preceded death.

Abstract of Necropsy.—Aorta extremely atheromatous, whitish or yellowish irregular elevations, fibrous-like on section, studding its internal surface. Above and in front of the right-most cusp of the aortic valve was an aneurysm of aorta, and a secondary one projected from this posteriorly. The aneurysm pressed against the commencement of the pulmonary artery; its walls were very atheromatous. The smaller secondary dilatation was somewhat distinct from the original one, but had a wider orifice than the narrow mouth of the latter, and its walls were thin, ulcerated, and with blood-stained *intima*, and below its neck was a patch of aortic calcification. The vessels of the chest and neck were gorged with dark blood. In the pericardium were eleven fluid ounces of dark sherry-coloured fluid. The cardiac muscle looked pale and somewhat fatty-like. The heart weighed 16oz.; the left ventricle was hypertrophied and dilated; all the other chambers were more or less dilated; of the auricles, the right more so than the left. The somewhat stenotic aortic valves were considerably diseased. Lungs congested and œdematous. In the abdomen was some ascitic fluid. Spleen, 15oz., soft. Liver, 51oz., fatty, passively congested in district of hepatic vein; old strong peri-hepatitic adhesions. Kidneys, 7 and 6oz., indurate, pale, yellowish. Cerebral leptomeninges congested, slightly opaque and thickened.

Case III.—A. M. Formerly the subject of monomania of persecutory and hypochondriacal type blended with some expansive delusions. Later says, "God and others speak to him in visions; internal voices cause him to speak and act without power of self-control.' His trachea is worked up and down. Has two personalities in his body; what he says has two meanings; has been sent to the asylum in mistake for someone else. A false nerve is worked on his body." He made treacherous homicidal attacks on attendants. Later, he became quiet, depressed, sombre, yet irritable and sullen; complained about his detention, was full of delusions as to ill-treatment and his bodily injuries and condition.

Abstract of Necropsy.—There were three aneurysms of the thoracic aorta. The first—a clot containing aneurysmal pouch, the size of a small orange, and about three and a-half inches long, one inch to the left of the origin of the left subclavian artery, and projecting leftwards from the junction of the arch with the descending aorta. The second, a lateral dilatation further down, chiefly of the left side of the aorta. The third, a pouch, projecting from the posterior wall, and eroding the bodies of the ninth and tenth dorsal vertebræ—a dilated pouch of very sharp contour, and abruptly limited. Aorta highly atheromatous, puckered, and nodular, some parts yellowish. The abdominal aorta was also atheromatous and pouched. The muscle substance of the heart was soft, friable; the aortic valves were opaque. The lungs were congested and slightly tubercular. Spleen, 7½oz., its capsule, and that of liver, irregularly thickened, &c. Kidneys red, granular, but of good size and weight.

Of the remaining cases of large thoracic aortic aneurysm little need be said. In some the mental modifications, apparently due to the effects of the aneurysm, were insufficiently marked, if present; in others the condition of physical disease was too much complicated for our present purpose.

CASE IV.—H. F. For several years before death, at the age of sixty, he had had thoracic aneurysm, and finally a heaving swelling on the upper part of the right side of the chest, its centre opposite the right third interspace near the sternum, the upper framework of the chest heaving perceptibly at each impulse of the heart and aneurysm. Right temporal artery large, prominent. Pulse and heart at times intermittent. Superficial veins of upper chest and upper limbs swollen, especially on left side. An abscess came under the jaw, and one on the dorsum of a foot. The urine had a marked deposit of alkaline phosphates. Later the pulse failed in left radial and brachial arteries. Finally, orthopnœa, profuse sweating, coldness chiefly in left upper limb; pulse rapid, feeble; left thorax veins very prominent; mucous râles over left chest; rapid increase of tumour.

An aneurysm of the aorta, nearly full of firm laminated clot, began about one and a-half inches above the aortic valve, projected forward, eroded the third and fourth right costal cartilages and adjoining parts, and was of the size of a hen's egg. Also, in the transverse arch was another and larger aneurysm, as big as a good-sized orange, which had pressed half-way through the sternum, and involved all the large arterial vessels of the neck at their origins, adhered closely to pleura and pericardium, and compressed the subclavian artery and vein, the latter of which contained a clot. The much dilated innominate artery sprang from the lowest and posterior part of the aneurysm. This second tumour pressed back upon the trachea and roots of lungs. A third aneurysm, one of the descending aorta, had eroded half through the bodies of the tenth and eleventh dorsal vertebræ and inter-vertebral substance. The entire thoracic aorta was studded with atheromatous and calcareous plates. Heart 12¼ ozs.; muscle pale; left ventricle hypertrophed; valves atheromatous.

Formerly the subject of exalted monomania (social and ambitious), this patient had greatly deteriorated in mental faculties; with his exalted ideas of rank and wealth were gradually increasing incoherence and childishness. Latterly, with the greater growth of aneurysm, he became restless, would suddenly start and move, and also became reserved, rarely speaking, but muttering irritably to himself.

Case V.—Here apathetic dementia had followed melancholia. The aneurysm of thoracic aorta was practically latent, both as regards physical (vital) symptoms and mental.

Case VI.—At first, mental depression, especially as to religious matters; improved, relapsed; finally, for years confused, rambling, incoherent, demented, but with paroxysmal excitement and always easily evoked irritability. But here the aneurysm of commencement of aorta was associated with granulo-fatty heart, and latent tubercular phthisis pulmonalis et abdominalis.

Case VII.—This too was a complicated case, organic brain disease being present as well as granular kidney, &c., and therefore unsuitable for our present purpose, like the preceding case, and like the next one, for in Case VIII. the aortic aneurysm was complicated by enormous hypertrophy and dilatation of heart, and marked aortic valve disease. The patient was expansive, maniacal (simulating exalted general paralysis), restless, irritable, difficult to manage, angry, resistive if not allowed to have his own way.

Abdominal Aortic Aneurysms.

Case IX.—Large aneurysm of abdominal aorta was complicated by aortic valve regurgitation. Old delusions as to plots, conspiracy, as to the effects upon others, at a distance, of the voices of himself and companions, as to mesmerism exercised upon him, or the hostile injurious influences exerted on him, still, by people whom he has not seen for years, and vivid hallucinations of sight and hearing were, latterly, when the aneurysm was marked, much replaced by delusions about personal injury and constraint, and by rare outbursts of sudden irritability, excitement and threats of destructiveness. Thus, at different times, he made statements such as that "he has people inside his skin, hurting him;" "his teeth have been knocked out by a man he did not see;" "something was thrust into his nape and caused the pain in his back;" "something like a hot iron came into his back;" "people come and look at him, and come into his face;" "gets his breath stopped from the thick air coming against him and stopping the suction of his nostrils;" "medicine has soaked all through his flesh and head;" "someone must have tight hold of him, as he can't have freedom of his legs;" "can't sleep, the book he reads plays on him, he is troubled and has no control over himself;" "gets strange smell; hears voice" (hallucinatory).

Heart, 13½ ozs., aortic cusps thickened, somewhat narrowed; aortic arch atheromatous, calcareous, rugose, somewhat dilated. Abdominal aorta opposite cœliac axis, dilated, and posteriorly opening into a large, and now ruptured, aneurysmal sac, which eroded the bodies of the second, third and fourth lumbar vertebræ, extended nearly into the spinal canal, and which adhered to the vertebral column and to the abdominal walls. A huge retro-peritoneal hæmatocele, the result of the sac's rupture, extended to the pelvis and surrounded the left kidney. Aorta atheromatous below, and other arteries so, also. Kidneys, 5 and 5½ ozs., and reported "healthy."

Case X.—Abdominal aneurysm, interfering with functions of intestines, and complicated by heart disease, chiefly aortic orifice stenosis and its effects on the heart.

At first, expansive delusions, chiefly on religious topics; later, with hypochondriacal and other ideas. "Has had poison, has vomited up all his bowels, is all sinews and membranes." Later, becoming incoherent, rambling in statement, inattentive, at times excited, noisy, destructive, and latterly uttering only a shrill unintelligible jargon.

Aneurysm of front wall of abdominal aorta, adherent to stomach, duodenum, pancreas, left supra-renal capsule; hæmorrhage into stomach. Left kidney, atrophied, granular, 1½ ozs.; right kidney, granular, 5½ ozs. Spleen, big, and its artery enormously dilated and tortuous.

Heart, 15½ ozs., stenosis, &c., at aortic orifice; great hypertrophy of left ventricle. Thoracic aorta extremely atheromatous, somewhat dilated.

Case XI.—Abdominal aneurysm; functional disorder distortion and displacement of intestines, and local peritonitis. Some hypertrophy of heart (left ventricle chiefly; right ventricle dilated).

At first restless, excited, owing to delusions of being poisoned, or annoyed by women. Later, restless, excited, reviling, contemptuous, in antipathy open; threatening, occasionally violent, obscene, blasphemous. Hallucinations of hearing and smell. Expansive tinge, *e.g.*, wrote to Queen, Government and newspapers. Latterly, more morose, depressed, suffering, pained in appearance and manner.

A large aneurysm of the upper part of the atheromatous calcareous abdominal aorta had ruptured at the level of the kidney, and, very much as in Case IX., a huge retro-peritoneal hæmatocele, enclosing the left kidney, had formed and extended to the pelvis. It and the aneurysm had caused extensive displacement and disease of the intestines. Kidneys $3\frac{1}{2}$ and $6\frac{1}{8}$ ozs., somewhat granular, artery of left one plugged by pale clot. Pancreas, indurate and hypertrophic.

Heart, $14\frac{1}{2}$ ozs. Left ventricle hypertrophied, right large valves altered and coarse, thoracic aorta atheromatous.

CHOREA,
WITH AN ACCOUNT OF THE MICROSCOPIC APPEARANCES IN TWO FATAL CASES.[1]

BY H. HANDFORD, M.D., M.R.C.P.,
Physician to the General Hospital, Nottingham.

BEFORE entering into a detailed description of the pathological histology of two fatal cases of acute choreic insanity described elsewhere (see under Clinical Cases in this number) by Mr. Evan Powell, to whom I am indebted for the material for examination, I should like to say that I do not approach this branch of the subject without having at the same time an adequate acquaintance with the clinical aspects of the disease. Chorea is very common in this town (Nottingham) and county, and during the past three and a-half years I have had twenty-four cases under my care in the General Hospital, where only the more severe or troublesome cases are admitted, the rest being treated as out-patients. And during the five years I was seeing out-patients daily, more than 100 cases of chorea came under my care. In looking through the record of admissions into the Nottingham General Hospital for the past thirteen years I find 154 cases of chorea have been under treatment, as recorded in Table I.

TABLE I

CASES OF CHOREA TREATED IN THE GENERAL HOSPITAL, NOTTINGHAM, FROM NOVEMBER 1ST, 1875, TO SEPTEMBER 30TH, 1888—A PERIOD OF ABOUT 13 YEARS.

Ages	3	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Over 20	Total
Males . ..	0	0	0	2	2	3	6	7	4	3	4	3	1	3	0	0	2	1	41
Females ..	1	1	2	1	5	8	6	4	13	8	10	11	12	5	5	3	2	5	102
Not Specified																			11
Total . .	1	1	2	3	7	11	12	11	17	11	14	14	13	8	5	3	4	6	154

Of these 154 cases four died, giving a mortality of 2·6 per cent. against 2 per cent. in the British Medical Association

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[1] Read before the Nottingham Medico-Chirurgical Society.

Collective Investigation Report. Four cases were pregnant, and one of these terminated fatally while in the hospital; the subsequent course of the others was unknown.

The following is a very short account of three of the fatal cases, for which I am indebted to my colleagues. Of the fourth case there is no record.

Case I.—Bridget McC., aged 17, had suffered from acute rheumatism about three or four months previously. Chorea commenced three weeks before her admission. The movements were extremely violent, and her temperature was 103°F. She died thirty-six hours after admission, and for the last twelve hours suffered from hyperpyrexia, attended by coma and cyanosis, with absence of the choreic movements. The temperature in the axilla rose from 105°F to 108°F. No post-mortem examination was allowed.

Case II.—Kate B., aged 25, unmarried, but in the fifth month of her first pregnancy, died of exhaustion two weeks after admission. The temperature varied from normal up to 102°F. Post-mortem refused.

Case III.—Lucy D., aged 12, convalescing from a mild attack of enteric fever, for which she had been in the fever block of the hospital for between two and three weeks. The first day she got up very slight choreic movements were noticed in the fingers. Next day the face and lower extremities were slightly affected. In three days a very severe attack was fully developed, and the temperature was 104°F. The patient died on the eighth day, the temperature varying for the last five days between 104° and 105·5°. A post-mortem examination was refused.

The proportion of males to females was as 1 to 2·41.

TABLE II.

B.M.A. Collective Investigation Record, 1882—1885.

Ages.	Males.	Females	Total.	Proportion of M. to F.
5 years & under	1	5	6	
6 to 10	46	102	148	1—2·21
11 „ 15	49	140	189	1—2·85
16 „ 20	15	56	71	1—3·73
Over 20	3	17	20	1—5·6
Total	114	320	434	1—2·8

TABLE III.

Nottingham General Hospital, 1875—1888.

Males.	Females.	Total.	Proportion of M. to F.
	2	2	
13	22	35	1—1·69
21	46	67	1—2·19
6	27	33	1—4·5
1	5	6	1—5
41	102	143	1—2·48

TABLE IV.

B.M.A. Collective Investigation and Nottingham General Hospital together.

	Males.	Females.	Total.	Proportion.
Ages 14 to 18 ...	35	122	157	1—3·5

The Nottingham Hospital cases give results which approximate closely with those obtained from the B.M.A. Collective Investigation record series. Age is the most important condition, inasmuch as 95·4 per cent. of all the cases occurred under the age of twenty. The influence of sex is very marked and very different at different ages. At all ages the proportion of males to females is in the B.M.A. Collective Investigation tables 1—2·8, and in the General Hospital Nottingham tables 1—2·48, showing a very close agreement. And since a very large number of the cases occur before the age of puberty it is evident that females are specially liable to chorea independently of any very definite sexual activity. But the not unfrequent association of hysteria with chorea, and the well-known influence of pregnancy, both in determining an attack and in inducing it to tend to a fatal termination, would lead us to suppose that sexual activity has some influence. And on looking at Tables II., III. and IV. it will be seen that the liability of the female sex as compared with the male rises steadily after puberty. If we divide all the cases in both series (B.M.A. Collective Investigation and General Hospital, Nottingham) into two divisions, viz.:—those under fifteen and those above, we see that in the former the proportion of males to females is 1—2·4 while in the latter it is 1—4·2, or little more than half.

TABLE V.

B.M.A. Collective Investigation and General Hospital, Nottingham, together.

	Males.	Females.	Total.	Proportion.
Under 15	130	317	447	1—2·4
Over 15	25	105	130	1—4·2
Total	155	422	577	1—2·72

With regard to the association of chorea with rheumatism, I have in a few cases seen definite articular rheumatism arise during recovery from chorea and *vice versâ*. But in the very large majority of cases I have not been able to find evidence of previous rheumatism *in the patient*, if "growing pains" and "vague rheumatic pains" be left out of account. A family history of rheumatism can generally be obtained, on enquiry, in this district from hospital patients, who attribute most pains to a rheumatic origin, and among whom rheumatism is in reality common.

Turning next to the condition of the heart, I feel that perhaps more attention than is its due has been paid to the presence or otherwise of murmurs, and too little to the other signs of affection of the heart. Some irregularity of rhythm has been attributed to irregularity of the respiration, and some murmurs have been supposed to be caused by a choreic affection of the papillary muscles. I have been accustomed to look upon many of the apical systolic murmurs as so-called "hæmic" murmurs—that is, in my opinion, murmurs due to dilatation, which in these cases is brought about by the mal-nutrition of anæmia and the excited cardiac action of chorea. It is generally acknowledged that the action of the heart in choreic patients presents peculiarities which are difficult to describe, but which are easily recognised. There is slight irregularity both in force and in rhythm, and the contraction is sharp and abrupt, and occasionally tumultuous. I cannot help thinking that this excited action is sufficient of itself to cause *mechanically*, in an ill-nourished heart, that peculiar form of endocarditis almost invariably found in fatal cases of chorea, viz., the small beaded vegetations round the margins of the valves, where they are liable to injury by contact. The small number and the diminutive size of the vegetations, and the absence of other signs of general endocarditis (as distinguished from limited valvulitis) in most of the fatal cases (which, of course, are the most severe), make me hesitate to consider endocarditis an essential part of the disease, and make me doubt whether the murmurs heard in non-fatal cases are often due to such endocarditis rather than to dilatation or other conditions. In fact I am inclined to look upon the endocarditis, when not rheumatic, as a comparatively trivial, mechanical complication, pretty constant in very severe cases, but contributing little, if at all, to the fatal termination. I have not met with optic neuritis or any complaints of impaired vision, and no systematic examination of the fundus has been carried out.

It is possible, therefore, that optic neuritis may have been overlooked, as it appears not necessarily to involve interference with sight.

The plantar reflex I have generally found diminished and sometimes absent, and I have attributed this largely to the very general coldness of the feet and impaired cutaneous circulation.

As all the cases of chorea that have come under my own care—about 130 in number—have belonged to the lower class, I have been accustomed to judge of their intelligence partly by the facility with which they answered ordinary questions and partly by the standard they had attained in the Board School examinations. The latter may, I think, be accepted as a fairly uniform test, and as a reliable gauge at any rate, of the degree of forcing to which their minds have been subjected, irrespective of its educational value. Estimated in this manner, I may say that I have rarely, if ever, met with a case of chorea in a child of school age that appeared stupid, or that was below the proper standard for its age. Usually, children that one would expect from their age to be in the third standard, would be in the fourth, fifth, or even sixth. This of course is quite separate from any impairment of the intellect which comes on in the course of the disease, and for which, I think, adequate reasons may be assigned. There seems to be a consensus of opinion that worry and anxiety are the most potent etiological factors in chorea. In children this is often brought about by intellectual forcing for examinations. The influence of anxiety and emotion is I think equally shewn in what might appear to be an exception, namely, the chorea of pregnancy. It is known that chorea occurs chiefly in first pregnancies, when emotion at an unusual condition, and anxiety in anticipation of the pains, discomforts and dangers of child-birth, and an intense wish to avoid any unfavourable course of life, are common. But still more does chorea occur in illegitimate pregnancies, when causes for anxiety are multiplied manifold.

None of the cases of chorea under my own care have proceeded to a fatal termination; and the only opportunities I have had of examining, post-mortem, the condition of the nervous system, have been in the two adult cases of acute choreic insanity, which Mr. Evan Powell kindly placed at my disposal, and of which he has just given the clinical history and the naked eye morbid anatomy. The first of the two cases I also had an opportunity of seeing during life, and the choreic condition was most manifest and typical. The brain in this case presented at the post-mortem examination a pink blush quite distinct to the naked eye. It was chiefly confined to the grey matter of the cortex, and could not be accounted for by the position of the body after death. As regards position and the opening of the cranial cavity first, the body was treated exactly as is the routine in scores of other cases, but the appearance of sections of the brain suggested by this soft diffused pink colour a capillary engorgement that is not usually seen. In the second case this pink blush was not so manifest. In each case the cord, and portions of the motor cortex and basal ganglia of the brain, were hardened in 2½ per cent. bichromate of potash solution. In the first case, unfortunately, the examination had to be delayed some months, and the specimens were in the meantime preserved in methylated spirit. This led to the production, to a typical degree, of the condition originally described as "miliary sclerosis," but now recognised as being in some way due to the action of alcohol. It is, I think, a gradual solution of the fatty substances, cerebrin lecithin and protagon, by the *prolonged*[1] action of the alcohol, which, if allowed to partially evaporate, deposits them again in globular areas, but not in any very definite crystalline form.

In the unaffected areas the sections are perfect, and the vascular changes are uninfluenced by the mode of hardening, and are sharply and clearly defined.

In the second case, in the majority of sections, alcohol was only used in the process of mounting in balsam, but in some instances the hardening of the tissue was completed by immersion in proof spirit for thirty-six hours, and strong methylated spirit for seventy-two hours, after washing out the excess of bichromate. There is no appreciable difference between the two series, except that the latter could be cut somewhat thinner. They were all cut after freezing. The staining agents have been ammonia carmine, alum carmine, logwood, bismark brown, and aniline blue black. I have found the latter and alum carmine the most useful. Altogether about two hundred sections have been examined. In sections of tissue that have been frozen, or subjected to very various processes in connection with hardening, staining, and mounting, I am always suspicious of slight alterations which appear to be of a degenerative nature and especially is this suspicion justifiable in the case of nervous tissue. But the changes I am about to describe are chiefly vascular and quite independent of the method of preparation.

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[1] **My experience has been that alcohol does not produce this change under several weeks or months.**

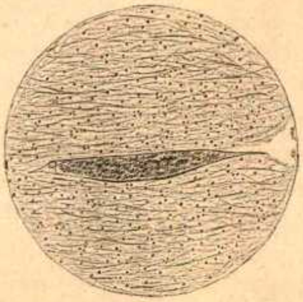

FIG. 1.—Thrombosed vessel shewn in Fig. 2 just above and to the right of the right anterior horn, magnified 250. Hæmorrhage into the lymph sheath is shewn by the size and shape, and by the absence of any sign of the vessel wall, which is hidden by the surrounding blood. Camera lucida drawing reduced ½.

I have not been able to detect any definite change in the nerve fibres or in the nerve cells, but there is, I think, generally an increase in the number of small round cells and leucocytes. But upon this point I should not like to speak positively. In both cases the engorgement of the vessels was striking, especially in the cord; and greater than could, I think, be accounted for by post-mortem hypostasis, inasmuch as I have only met with a similar degree in the medulla in a case of hydrophobia. In many instances there was nothing to suggest that the thrombosis had taken place during life, for the blood corpuscles were quite distinct, and there was no surrounding inflammatory condition. But in others there were hæmorrhages into the lymph sheath (Fig. 1), completely surrounding and hiding the walls of the vessel itself. These must have occurred during life, as also must the very numerous small hæmorrhages which were found most abundantly in the cervical region, but also in other parts of the cord, in the pons, and less frequently in the motor cortex. (Figs. 2, 3, 4, and 5.)

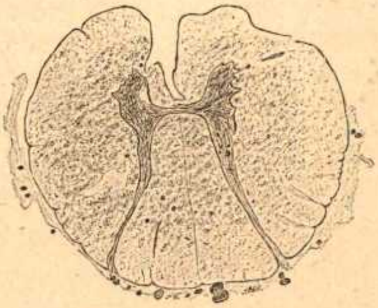

FIG. 2.—Dorsal cord. Thrombosis of vessels, especially near the anterior horn on the right side; thrombosed vessels are represented black. The adherent pia mater is especially vascular. Alum carmine staining. Zeiss ob. ai, oc. i. magnified about twelve diameters. Camera lucida drawing reduced ⅓.

In many instances they were found in the grey matter, especially of the anterior horns, where they must have interfered with the nutrition and function of the ganglion cells. Such hæmorrhages may account for the loss of power in the paralytic forms of chorea. A hæmorrhage which had led to destruction of tissue had taken place in the cervical region on each side of the central canal in the commissure in the position of the vertical veins. (Fig. 6). That this had taken place during life was shown by the darker staining of the margins of the spaces from slight cellular infiltration, by the torn-up nerve fibres, and by the presence of coloured blood corpuscles all around the margins of the spaces. The position of these comparatively large vessels (for there appear to be several enclosed in one sheath) on each side the commissure, and the entrance of arteries from the anterior fissure through the commissure form special sources of danger. It is possible that in this way centripetal impulses may be interfered with and some degree of incoordination accounted for. In this case the commissure was entirely interrupted.

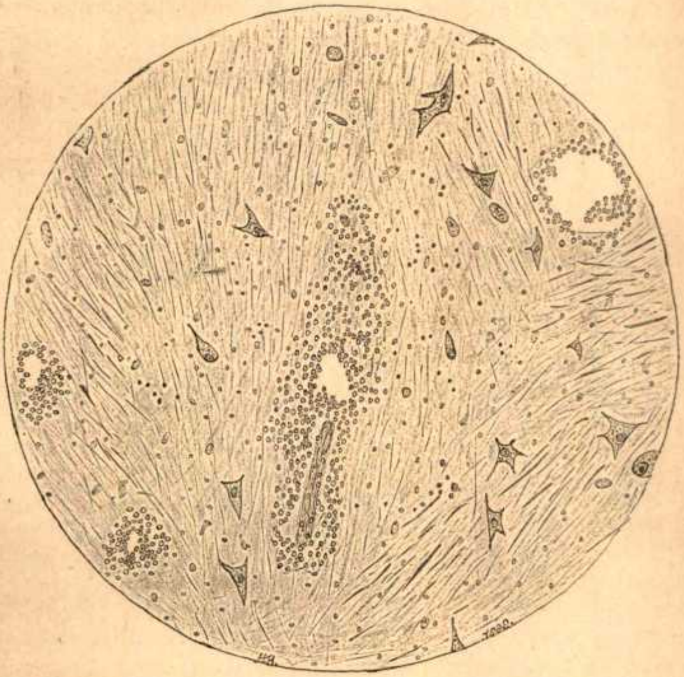

FIG. 3.—Cervical cord. Four hæmorrhages in the right anterior horn, causing destruction of nervous tissue, and leaving spaces where the clots have fallen out. A vessel is imperfectly seen in the largest hæmorrhage. Aniline blue black staining. Magnified 300 diameters. Chief outlines traced with camera lucida.

I am aware that it is stated that "the fibres of the anterior commissure are displaced by the vessels, and hence, in section, the commissure often appears to be interrupted." I can confirm this from my own observation of sections from very various subjects. But not only do the vessels entering (and leaving?) the cord from the anterior fissure displace the fibres of the anterior commissure, but they seem specially liable to dilatation and rupture. From their size and direct connection with the vessels of the pia mater they early feel increases of pressure; and from their being surrounded not directly by the nervous tissue but by a distinct fibrous sheath enclosing an unusually large lymph space, they appear to be accustomed to vary greatly in size. I have not before met with such a complete interruption of the whole commissure, anterior and posterior, as is represented in Fig. 6, or such a definite appearance of the ploughing up of the surrounding tissue by the blood which had escaped from the vessels. But the nearest approach to it was in the lumbar region of the cord of a nine months' fœtus which,

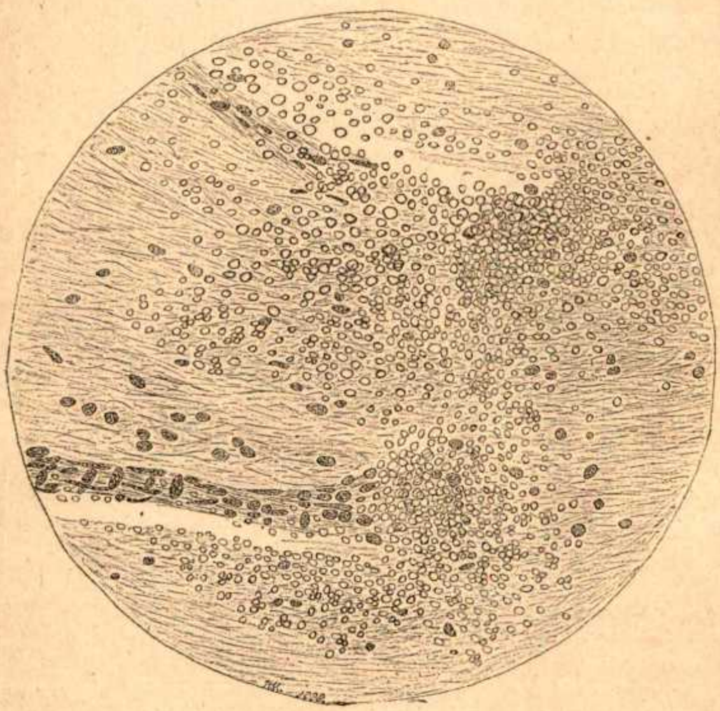

FIG. 4.—Cervical cord. Hæmorrhage from small vessels in the left anterior horn. In the upper of the two hæmorrhages it is difficult to trace the ruptured vessel. Alum carmine staining. Magnified 480 diameters. Outlines traced with camera lucida.

owing to the attachment of a large thyroid dermoid tumour to the sacral region, was asphyxiated by the difficult and prolonged mechanical delivery. Here were present all the conditions of violence, and increased blood pressure from asphyxia favourable to vascular rupture.

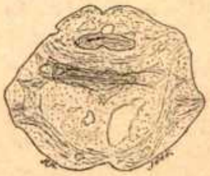

FIG. 5.—Transverse section of the pons about the level of the fifth nerve. Two-thirds natural size. Hæmorrhages into the deep transverse fibres and into the reticular formation.[1] Outline traced with camera lucida.

Fig. 7 shows a vessel in the pons with a thrombus, or an incomplete adherent embolus attached to the side of the vessel which is dilated before it and somewhat contracted

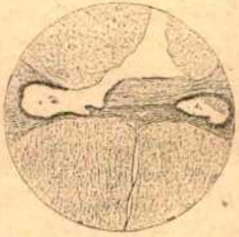

FIG. 6.—Cervical cord. Two large hæmorrhages, *a* and *b*, in the commissure. The nerve fibres are torn up and lie in all directions at the margins of the spaces from which the clots have fallen. That the spaces were formed during life, and not from imperfect hardening, or from manipulation of the section is shown by: (1) The perfect hardening of the rest of the section. (2) The darker staining of the margins of the spaces. (3) The cellular infiltration of the margins. (4) By a considerable number of coloured corpuscles still adhering to the margins. Aniline blue black staining. Magnified 85 diameters. Camera lucida drawing reduced $\frac{2}{3}$.

beyond it. The whole vessel was full of blood, showing the obstruction to be incomplete. This was the only appearance suggestive of embolism that I succeeded in finding.

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[1] An almost exactly similar figure is given in Carswell's 'Pathological Anatomy,' Plate I. "On Cerebral Hæmorrhage," Fig. 4.

In the pons about the level of the fifth nerve, were two hæmorrhages, represented in Fig. 5. They were in the deep transverse fibres and in the reticular formation, were not quite recent, and showed general blood-staining, but the individual corpuscles could not be detected and seem to have disintegrated. They seem rather too high to have affected the respiratory centre, and neither sufficiently extensive nor recent to have been the immediate cause of death. No definite alterations were detected in the basal ganglia, and the vascular changes were less marked in the motor cortex than in the spinal cord.

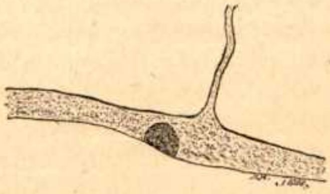

FIG. 7.— Thrombus, or incomplete adherent embolus attached to the side of the vessel, which is dilated before it and somewhat contracted beyond. The whole vessel was full of blood, showing the obstruction to be incomplete. From the pons, magnified 400 diameters. Outline traced with camera lucida.

Finally, I think that the very numerous hæmorrhages from small vessels as well as capillaries, the thromboses and the general dilatation of vessels afford evidence of a very unusual degree of vascular engorgement of the nervous centres during life; and that this is the essential feature of chorea.

Dr. Sturges has defined chorea as "an exaggerated fidgetiness," and Dr. Dickenson has expressed the opinion that the condition is due to "a widely-spread hyperæmia of the nervous system." These two statements, I think, express a very large part of the truth about chorea. Apart from the general nervous type, malnutrition and overwork (the latter being an upsetting of the proper balance between work, rest and nutrition) are the natural factors of fidgetiness.

In otherwise healthy persons, overwork produces a condition in which the subject is easily startled by slight noises, is worried by persistent sounds, has trembling of the hands and impaired sleep. The natural hyperæmia of the brain necessary for active work does not subside when the time for rest comes round and prevents sleep. In addition to this, though neither choreic movements nor inco-ordination may arise, manual dexterity may almost completely fail. Actions are performed hurriedly, but clumsily and inaccurately, and objects are readily dropped or broken.

I remember some years ago a distinguished Professor of Physical Science and Engineering, who, though usually an exceedingly dexterous and skilful mechanician, when overburdened by a vast amount of examination work involving late hours, became so clumsy that his demonstrations on lathe work became positively dangerous from his letting tools slip and be whirled away. The progress from this condition to chorea is not a great step.

I may further mention the not infrequent muscular twitchings present during "nervous" conditions from overwork, which disappear with rest and improved health and nutrition. For example, the twitching of the orbicularis palpebrarum after fatigue.

Emotion is from clinical experience a recognised factor in the etiology of chorea. Emotion is a very definite cause of vascular excitement, as shown in the form of cardiac palpitation, and blushing of the skin. It is no great assumption to suppose that the parts we cannot see or feel blush and become hyperæmic also.

I think that there is abundant evidence to show that "a widely-spread hyperæmia of the nervous system" is capable of producing all the active symptoms of chorea, and that the consequent thromboses and hæmorrhages will explain some, if not all, of the inco-ordination and motor weakness, and that this view of the etiology of chorea is supported by the pathological evidence.

The share that is taken by the different portions of the nervous system in the production of chorea is still an open question. I am inclined to look upon the milder cases as resulting from an affection of the cord—the lowest evolutionary level; and the severe ones associated with mental disturbance or definite insanity as resulting when the same process spreads to the cerebral cortex—the highest evolutionary level.

Chorea in the dog is, we know, due to some affection of the cord. But it is argued that canine chorea is not the same disease as human chorea. It is at any rate a very closely allied one.

The chief reasons alleged against the supposition that chorea is an affection of the cord are the following:—

(1) That the choreic movements cease during sleep, when the excitability of the cord is normally increased. It is very doubtful whether the movements do always cease completely during sleep, although undoubtedly that is the rule.

(2) The limitation of the movements to one side or to one limb. Strict limitation of the movements to the whole of one side of the body, or to the arm and leg on the same side is very rare, at any rate for more than a very short period; and the amount of unilateral limitation usually observed is equally seen in anterior polio myelitis.

It is quite as difficult to suppose a vascular change, (excepting destructive ones such as hæmorrhage) to be limited to a small portion of the cerebral cortex.

But the strongest ground of all for thinking that the morbid change is not primarily in the motor cortex, consists in the fact that that region can generally be shewn to be capable of working properly and of controlling the irregular movements. No fact can be more undoubted than that choreic movements are to some extent under the influence of volition. In mild or moderate cases the movements can be entirely checked for short periods at a time by an effort of the will.

Clinical Cases.

I.—ABSCESS OF THE PARACENTRAL LOBULE BURSTING INTO THE LATERAL VENTRICLE.

II.—ANEURISM FOLLOWING EMBOLISM OF THE ANTERIOR CEREBRAL RUPTURE INTO THE RIGHT LATERAL VENTRICLE.

BY FREDERICK W. MOTT, M.D.

I.

E. F., æt. 42, was admitted into Charing Cross Hospital for hemiplegia on December 16th, 1888. He was placed under the care of Dr. Green.

His family history was unimportant, save that his sister had died of paralysis. He had enjoyed fairly good health up to a year ago, when he met with an accident. He worked as a mason, and twelve months ago he fell off the scaffolding, and was admitted into the hospital under Mr. Barwell. He was insensible at the time he was brought to the hospital but there is no other mention of head injury in the notes, and the case is described as one of fractured ribs and sternum with surgical emphysema. He recovered, although he had a sharp attack of pleurisy, and he was able to resume his work after a few months.

History of Present Illness.—On December 3rd he was attacked with a severe headache, which continued, with exacerbations, throughout the week.

On December 10th at 7.30 a.m. while at work he felt a severe stabbing pain at a spot situated one inch inside the right nipple. The pain rapidly, and, as far as he could tell, extended to the right arm and leg, and the right side of the face. He had time to sit down, and then the affected limbs began to jerk convulsively, and for the time being he lost all control over them. The fit lasted about ten minutes, and he did not lose consciousness. He did not bite his tongue or pass his water involuntarily. As soon as the fit was over he felt perfectly well and resumed his occupation again. Nothing further happened till the next morning, December 11th, at 5 o'clock, when he was awakened by severe pain, and then he had another fit of a similar character to the one already described. On this occasion he lost consciousness, and the bladder was emptied involuntarily. He did not bite his tongue. The fit lasted about ten minutes; he recovered completely, but he was greatly alarmed. He went to his work at 10 o'clock, and had another fit which he states was severer than any previous one. At 12 o'clock he came to the hospital and was seen by the house physician who gave him some medicine. He did not have another fit till the next day, December 12th. He recovered from this fit, but he noticed that his limbs were numb and his hands felt as if covered. He continued in this condition till December 16th, when at 12 o'clock, while filling his pipe his hand suddenly dropped and he found he had lost power in his right arm and leg and right side of the face. Sensation remained normal. He was brought to the hospital and admitted.

Condition on admission, December 17th, 1888.—The patient is lying in bed on his back; he can answer questions very well, and gave the above clear history of his present illness. He has no loss of memory but his speech is a little thicker than before.

Arm.—He can move the fingers of his right arm a little, and he can move the forearm also slightly; yesterday the paralysis of the arm and fingers was complete. No loss of sensation.

Leg.—He can raise his foot from the bed but there is great loss of power in the lower limbs. Sensation is normal.

Trunk.—No loss of sensation or power in the trunk muscles.

Face.—There is slight weakness of the right angle of the mouth. He can whistle. On admission, the right side of his face was paralysed. The tongue seems to incline very slightly to the right side, when protruded. He states that the mouth feels numb at the angle.

There was increased knee jerk, and slight ankle clonus in the right side.

Eyes.—No diplopia, pupils equal, field of vision not diminished. No loss of power of movement. Ophthalmoscopic examination revealed slight optic neuritis on both sides, without swelling of the disc.

There is pain over the vertex of the skull; and on manipulation, pressure causes extreme pain and tenderness over the situation of the junction of the fissure of Rolando with the longitudinal fissure. Temperature normal. Bowels acted once to-day.

December 19th.—Temperature 100°, condition otherwise much the same. He was seen by Dr. Green; as syphilis could not be excluded (although there was no history of it), he was put upon pot. iodid. gr. x. and pot. bromid. gr. x., three times a day.

December 20th.—Patient is unable to move the fingers or arm. Temperature normal. Urine normal. Pot. iod. increased to 15 grains.

December 21st.—Patient vomited this morning, and twice during the night. He feels much worse than he did yesterday. Temperature normal. An ice bag was applied to the head.

December 22nd.—Patient feels much worse. The breath has a peculiarly offensive odour. The tongue now protrudes to the right, and the facial paralysis is more marked. He has vomited once since yesterday. Complains of headache still. Temperature normal.

December 23rd.—Facial paralysis more marked. Headache more severe. Speech slow, drawling and indistinct. No movement in arm or leg. Slight vomiting. Pulse slow, 60, hard. Respiration 16.

December 24th.—He can hardly speak; breath very foul. Tongue furred. He takes his food well. Paralysis of face increased. Vomited once yesterday after his medicine. Sleeps very little, and cries out in the night. Pulse 64.

December 25th.—Patient is still semi-conscious, and can recognise his relatives; takes no food, and passes his urine and fæces under him.

December 26th.—He became rather suddenly quite unconscious and cannot now be roused. The left pupil reacts more readily to light than the right, but they are equal. Pulse 80. The temperature for the last two days has been sub-normal. From this condition he did not recover, death occurring twelve hours after he became unconscious.

Autopsy made eleven hours after death.—On removal of the scalp no trace of injury of the skull was discovered, nor on removal of skull cap. After slitting up the dura mater along the longitudinal sinus, it was reflected from the surface of the brain in the usual way. The convolutions about the fissure of Rolando of the left hemisphere are flattened, and softer, and wider than on the right side. Over the paracentral lobule there is a little elevation of the arachnoid, about the size of a sixpence, and of a greenish colour. In this region an elastic fluctuation can be made out. The convolutions which were softened were the upper portions of the ascending parietal and frontal convolutions, the paracentral lobule and the adjacent supra marginal and angular gyri. The brain was removed, and while separating the hemispheres very carefully, pus burst through the portion of the corpus callosum forming the roof of the left lateral ventricle, a sinus was found to extend from the left lateral ventricle into the abscess cavity. The abscess cavity was about the size of a large chestnut; it involved the whole of the structures of the paracentral lobule, and it contained a quantity of thick greenish pus not offensive in smell. The direction which the abscess took was downwards and inwards, so that it did not seriously involve the supra marginal convolution; although it undermined it completely. The wall of the abscess cavity was firm and vascular, but there was no very distinct pyogenic membrane. At its thinnest part it measured a quarter of an inch; this was situated at the upper portion of the ascending parietal convolution, close to the greenish elevation of the arachnoid.

As far as could be judged there was about ʒij. of pus. Microscopical examination of the wall of the abscess cavity shewed most internally leucocytes and dilated vessels, and external to this degenerated nerve fibres with leucocytes.

No cause was found to account for this abscess. The vessels of the brain were not diseased, and the heart and its valves were healthy.

Lungs:—*Right lung* was universally adherent, the adhesions being old and tough. There were old cicatrices in the lower lobe. The left lung contained numerous cheesy nodules at the apex; otherwise fairly healthy.

The remaining organs were healthy, except slight interstitial change of the kidneys.

Commentary.—This case is interesting in many ways: firstly from a general pathological point of view, and secondly in connection with special cerebral localisation.

I must confess that it was my opinion the case was one of cerebral tumour, and abscess never occurred to me, although I felt sure that it was a case in which a surgeon might have been most advantageously called in. Dr. Green, under whose care the patient was, felt very properly that before any operative measures were proceeded with, the patient should be put upon anti-syphilitic treatment. For a few days the patient's condition seemed to improve; but at Christmas time, and while Dr. Green was out of town, the patient somewhat suddenly became worse, and he died on Christmas Day. On account of the evident signs of irritation, followed by destruction of the portion of the motor area corresponding to the paracentral lobule and adjacent structures, the surgeon would have had no difficulty in trephining over the exact spot, and on exposing the brain in that region he would have met with the elastic fluctuating abscess. The case would have been more favourable for operation than a tumour, because if the latter had produced the symptoms, it would have meant a general infiltration into the structures surrounding the paracentral lobule in order to have given rise to the symptoms. *The diagnosis* lay between (1) tumour, (2) gumma, (3) acute cerebral abscess the result of infective embolism, and (4) latent cerebral abscess, from injury, becoming acute.

The only medical treatment that is likely to do any good in a case of cerebral disease having symptoms like the above is based upon the presumption that it may be syphilitic, and a case that was in the hospital three years back with symptoms very like the early stages of the present case yielded entirely to anti-syphilitic remedies, the patient being discharged cured. Of course it may be urged that the man had no symptoms and gave no history of syphilis; but then are these not the cases which so often present syphilitic lesions of the nervous system? Although the man had had a severe injury a year previously, yet there were no head symptoms except insensibility at the time (although it is possible they may have been overlooked on account of his other severe injuries), still there might have been some morbid process which, as frequently happens in these cases, remains latent for a time, and these symptoms manifest themselves and run an acute course. In many of these cases which have been reported, no injury of bone has been recorded and the cause has not been determined. It is possible that it was of an embolic nature. Bronchiectasis, gangrene of the lung, as I myself have seen, may give rise to cerebral abscess, but the lesions in the lung, found at the post-mortem, were hardly of the nature to account for the abscess being formed in this way. Moreover, although single abscesses may occur in cases of this kind, yet they are often multiple, and being infective would, I presume, give rise to pyrexia; but in the present case the temperature only once ran above normal, and then only reached 100°. Estimating the value of all these facts, I think the probabilities are, the case was one of traumatic origin, remaining latent for a year.

Some of the symptoms, especially at the onset of the disease, are of interest. Firstly, the aura which ushered in the first two or three *fits* of convulsions. The patient, in a very intelligent manner, described the warning which he received by a pain situated one inch to the inside of the right nipple, and then radiated from this spot to the right arm and leg and side of the face, followed after a short interval by convulsions.

In connection with cerebral localisation, this case seems to support strongly Dr. Broadbent's views regarding bilateral association, because the marginal convolution could not have created the morbid process entirely, and if the cortical portion of this gyrus did escape, I cannot conceive how the fibres escaped; moreover, the aura commencing in the right breast as it did distinctly pointed to this area of the brain being affected. In a conversation with Mr. Victor Horsley, who has proved by his experiments on monkeys that stimulation of the marginal convolution is followed by movement of the trunk muscles, he asked me a very pertinent question with regard to the proof that the trunk muscles were unaffected—Had I taken any special means of determining this fact, beyond watching the movement of the abdomen and chest of the patient while lying on his back in bed? I confessed I had not sat the patient up in bed and noticed the action of the recti. He stated that he had been unable to notice the paralysis in monkeys unless he took special means. So that although for my own part I do not believe the trunk muscles were paralysed, yet this part of the case is necessarily robbed of much of its scientific value because I cannot be sure of this point, and as cases of this nature occur so very seldom, it is a matter of regret to me that I did not take the precautions required to investigate the matter in a more convincing manner.

II.

W. S., æt. fourteen, was admitted into the accident room of Charing Cross Hospital, July 16th, 1889. He was brought by the police in an unconscious condition. He was suffering from great dyspnœa. The right pupil was larger than the left, and neither reacted to light. The corneal reflexes were absent. Patient still had some power in his limbs. There were no physical signs to indicate any affection in the chest, and it was thought possible that he had fallen down and fractured the base of his skull. Nothing could be made out to indicate any injury. Twenty minutes after admission he died.

At the autopsy some recent vegetations were found on the aortic valves. The left ventricle was dilated. There were three recent infarcts in the spleen, perhaps a week old at the outside. There was nothing noteworthy in any of the other viscera. All the organs were in an advanced state of decomposition owing to the order from the coroner being delayed.

On removing the skull-cap and slitting up the dura, a thin layer of sub-arachnoid hæmorrhage was found all over the anterior portion of the right cerebral hemisphere, and all the convolutions appeared flattened.

On separating the hemispheres the corpus callosum was seen bulged upwards, and the anterior portion was covered with blood clot firmly adherent. On opening the lateral ventricles they were found filled with clot, which on removal weighed one-and-a-half ounces. The vessels were now very carefully examined and washed, and the anterior portion of the corpus callosum with a portion of blood clot was found firmly adherent to the right anterior cerebral artery, one inch behind where it curves over the corpus callosum. On further careful washing a small aneurism about the size of a split pea and similar in shape, was found connected with the right anterior cerebral artery. This had become adherent by an inflammatory process to the roof of the lateral ventricle and rupture had taken place into it.

Comments.—This case is one of considerable importance, both clinically and pathologically. A diagnosis was rendered almost impossible owing to the absence of history; the sudden onset, together with the absence of any signs of injury might have suggested embolism or rupture of an aneurism, had it been possible to have recognised the heart condition, but the valvular disease was very slight and not enough to lead to incompetence.

The *post mortem* revealed the pathology of this case. Slight endocarditis, with vegetations on the aortic valves, and the recent infarction of the spleen, showed that portions had been detached; one had lodged in the right anterior cerebral, blocking it and giving rise to sub-arachnoid hæmorrhage in the area of distribution of this vessel. At the point occluded inflammation had occurred leading to adhesion and some destruction of the corpus callosum, the formation of a small aneurism and eventually a rupture into the right lateral ventricle. According to Dr. Gowers, from the statistics of 154 cases, the proportion of aneurism of the anterior cerebral to other arteries of the brain taken together is one to eleven.

CHOREA INSANIENS.

BY WILLIAM GAY, M.D., M.R.C.P.

There is generally some evidence or other of psychical disturbance in the course of chorea, but its coincidence with profound mental aberration amounting to insanity, is of sufficient rarity and interest to merit a passing attention. The following example of it is worthy of remark chiefly on account of the youth of the patient and the somewhat ambiguous signs of chorea.

J. E.,[1] æt. seven years, came under my notice at Great Ormond Street Hospital for Sick Children on August 23rd, 1886. There was no hereditary history of rheumatism, nor of any neurosis. The eldest of four children, the patient was always nervous and excitable—unlike the others, who were quite strong and healthy. There is marked evidence of old rickets, but no history of convulsions, laryngismus, enuresis or somnambulism. He is subject to night-terrors. His condition is ascribed by his parents to a fright caused by his incarceration in a dark cellar as a punishment at school (the infants' department of the Board School). This happened about ten weeks before he was brought to Great Ormond Street, and almost immediately it was noticed that he began to lie about and to be unable to make any exertion. He soon became unable to walk, and at last was so completely paralysed that he could move none of his extremities, nor even sit up. At no time did his parents observe any muscular twitchings. From the very commencement the patient's restlessness and tendency to emotional outbursts were intensified, and he became mischievous and bad-tempered, altogether unlike his former self. At times during the fortnight before I saw him he was wildly delirious, and proved so unmanageable that on three or four occasions he had to be strapped to his bed.

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[1] I am indebted to the kindness of Dr. Abercrombie for permission to publish this case.

Present Condition.—He is an emaciated boy with a complete want of expression, which gives him a very demented appearance. There is an occasional twitching of one of the angles of his mouth, and he is constantly dribbling. On asking him to put his arms out straight in front of him, he did so in that circuitous and irregular manner so characteristic of chorea. His tongue was also jerked out for me to see in the spasmodic way peculiar to choreics. When at rest no movements betrayed the presence of chorea, beyond the infrequent twitchings of the mouth. His limbs, and indeed the whole of his muscular apparatus, seemed in a paretic condition, and every movement was weak and ill-sustained. In this respect he had shewn considerable improvement of late for, according to his parent's account, he had at one time been unable to move at all. The improvement, however, in the physical symptoms was accompanied with an exaggeration of the psychical. At the time of his examination he was generally quiet and obedient, but now and then rambled incoherently. There was no history of rheumatism, no heart-disease, nor were any subcutaneous nodules present. He was sent into hospital in the hope he would be benefited by the discipline and restraint of hospital life, but he proved so wild and intractable that he was sent home the next day.

Happily for the further observation of the case, he was again brought to the hospital, and on August 26th I note that he is now incessantly chattering at such a rate that his words are unintelligible, and that this is only varied by emotional outbursts. He is unsteady with his legs together and eyes shut. Knee jerks equal and normal. For some days he has vomited in the early morning before taking food, and from the commencement of his illness has complained of headache. Optic discs normal. He was given cod liver oil, iron and arsenic, and a fortnight later my note says: "tremendous change, is quiet and rational, no choreic movements. Walks well, and there has been a great increase of power everywhere. Sleeps well." I saw the boy from time to time afterwards. He became quite lively and intelligent, and lost all traces of his former severe illness.

The evidence in favour of the essentially choreic nature of this case is not overwhelming. There was no rheumatism, no heart disease, no insanity of the muscles, but rather a paralysis. Motor weakness however is strikingly apparent in many cases of undoubted chorea, and I am inclined to think is nearly always present in a greater or lesser degree, and that not infrequently it is in inverse proportion to the amount of irregular movement. At any rate when the paralysis attains an inordinate degree, the real nature of the case may only be revealed by slight and infrequent twitchings in one part or another of the body. Such then was probably the state of affairs existing in the case of the boy recorded above. At one time he was so weak that he could neither move his extremities nor sit up, a condition which is an almost exact reproduction of a case noted by West.[1] The twitchings in J. E. were so slight that they were altogether unrecognised by his parents, who were acquainted with chorea, but were sufficient, especially when taken in conjunction with the sudden protrusion of the tongue and the irregular efforts to extend the arms, to establish the diagnosis. The chief interest of the case however is centred about the mental phenomena, which were marked from the very onset of the illness, but attained their greatest degree some weeks afterwards. The boy presented the appearance of dementia, but from his parents' account must have been subject to maniacal outbursts of considerable violence. There was no history of delusions, nor was there ever any incontinence of urine or fæces.

It is a matter of common observation, and has been particularly noticed by Watson, Radcliffe and Hillier, that chorea especially occurs in those children who are popularly known as "nervous." They are restless, irritable, "easily stirred by new ideas and sudden emotions, and pass readily and upon slight occasion from one mood to another" (Watson). Such children are essentially impressionable and easily affected by external influences. Upon them fright produces an effect out of all proportion to that which it would have upon their more phlegmatic brethren, and the influence of fright as an exciting cause of chorea is indubitable. It may be noted in this respect that in certain rare cases a great nervous shock occurring in the course of chorea has resulted in its cure. An excellent example of this is mentioned by Dr. Hughes. A girl suffering from her second choreic attack was on her way to Guy's Hospital for re-admission. As she was crossing London Bridge she saw somebody knocked down and run over. Before she reached the hospital her disease was gone. It is also worthy of remark, without insisting too much upon its value, that in the collective investigation report upon chorea, seventy-one cases (or 16 per cent.) were ascribed to mental overwork.

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[1] 'Diseases of Infancy and Childhood,' sixth edition, p. 224.

The characteristic motor phenomena are preceded frequently (Trousseau indeed asserts in the great majority of cases), by prodromal symptoms, all pointing to an enfeeblement of the intellectual faculties. The temper is changeable, the memory less retentive, there is a lack of concentration, and the child becomes capricious and moody. These symptoms tend to increase with the development of the motor affection, but so striking does the latter become that the mental condition is liable to be passed unnoticed or to be attributed to the severity of the movements. Hence the paralytic forms afford the best field for the observation of the mental aspect of the disease, for in them the movements are not sufficiently disorderly to mask other symptoms that may be present. Probably in every case there is some evidence or other of psychical disturbance which however bears no necessary proportion to the degree of the motor affection. In nearly every case there is a want of emotional restraint. Choreics are readily excited to peals of peculiarly explosive laughter, often for no very apparent reason, and are as easily moved to tears. They become in fact what is commonly known as hysterical. There is frequently a blunting of the intellectual faculties, and the patient is dull and obtuse. In some cases the memory is profoundly affected and in others the mental condition is described as "strange" or "peculiar." Such severe symptoms are not common and generally subside *pari passu* with the improvement in the motor disturbances, but cases are occasionally reported in which a persistent mental defect has remained.

Mania, as it occurs in the course of chorea, seems therefore to consist of a great exaggeration of the mental symptoms which are so frequently present in varying degrees. It is extremely uncommon before the age of puberty, and more generally occurs in girls and pregnant women. Of twenty-three cases of maniacal chorea, collected from various sources, five only occurred in males (from fourteen to nineteen years of age), and eighteen in females (from fourteen to twenty-five years of age), nine of whom were known to be pregnant. The choreic movements may be extreme, or so slight that the nature of the affection might easily be missed. In one case the mental symptoms, following a severe fright, anticipated the development of the motor disorder by eight days. In four others the psychical troubles became exaggerated as the choreic movements declined. In the remaining cases the motor and mental phenomena were coincident in their development. Delusions and hallucinations were present in several instances; in two cases there was a suicidal disposition, and in another a condition of melancholia succeeded several semi-maniacal attacks. Complete recovery (in two or three instances after a sojourn in an asylum) resulted in ten cases, permanent weak-mindedness in one, death in eight, and in four cases the patients were lost sight of in asylums. The absence of heart disease and rheumatism was noted in eight of the non-fatal cases, and their presence in two. Vegetations were present on the valves in four of the fatal cases and in one there was a slight thickening of one of the cusps of the mitral. In the remaining cases, details on these points were wanting. The actual cause of death is to be attributed rather to the exhaustion resulting from the mental and physical breakdown than to the condition of the heart. The fatal event sometimes ensues with great rapidity. In one extreme case the symptoms ran a course of only two days, and in another of six days, before death occurred. Much more generally, the disease is prolonged over some weeks.

The frequency of psychical disturbances in the course of chorea, even though they be as a rule comparatively slight, conclusively shows that a purely motor or even sensori-motor pathology does not cover the whole of the case. They afford indeed a certain amount of evidence in favour of the functional nature of the disease. The fact that those children are most generally affected whose nervous systems are mobile and most delicately balanced, goes hand in hand with the statistical observation that three times as many females as males suffer from chorea. It is difficult to reconcile this with the organic conception of the disease, nor is it easy to understand in what way a shower of minute emboli, even though they were able to produce the motor disorder, could cause in many cases those prodromal symptoms, already noticed, pointing to psychical disturbance. Again, the influence of fright, or of some great emotion as an exciting cause cannot be doubted in a great number of cases, but it hardly appears possible to make this harmonise with the coincident disengagement of emboli from the heart. The cure of Dr. Hughes' case by fright accords with the occasional behaviour of those functional affections with which chorea seems most nearly related, but upon the organic hypothesis it is as difficult to account for as certain cases recorded by Radcliffe and Rilliet and Barthez, which were cured by the onset of an exanthem. It may be urged that chorea is in some cases of organic, and in others of functional, origin, but it does not seem reasonable for the sake of a theory to dispute the entity of a disease whose onset, symptoms and course are so well defined as chorea. The psychical symptoms, like the motor, are in the main characterised by want of control and paralysis—there is a loss of emotional restraint and a blunting of the intellectual faculties. These on the whole would be more readily explained upon a functional hypothesis, and their presence suggests that, at least in some cases, the level at which some of the symptoms of chorea occur is considerably higher than the corpora striata.

TWO FATAL CASES OF ACUTE CHOREA, WITH INSANITY.

BY EVAN POWELL,

Medical Superintendent of the Nottingham Borough Asylum.

CASE I.—Albert S., æt. nineteen, single, was admitted into the Nottingham Borough Asylum on the 16th September, 1885, with the following *family history*. Father died of phthisis; mother probably syphilitic, and had suffered from acute rheumatism when sixteen years old.

Personal History.—Had a little rheumatism fifteen months ago in his shoulders, and three weeks ago had violent pain in his feet, also pain and stiffness in his calves; these symptoms increased rapidly until he was unable to stand, but after ten days' rest and treatment he got better and resumed work. Three days later he began to twitch in his left arm and left side of face; the movements gradually increased and in three days spread to the right side and became general. His mind now became affected; he got maniacal and developed delusions and hallucinations. He was of a nervous and excitable disposition, and just before this attack he had been subjected to some mental strain.

On admission he was found to be suffering from acute choreic movements, apparently of all his voluntary muscles, was unable to stand without support, his features were much contorted, he looked wild and haggard, and was unable to utter an intelligible word; tongue dry and fissured, lips cracked, bleeding and covered with sordes. Pupils equal and active to light. Pulse strong, but could not be counted. Temperature normal. No swelling or tenderness of any joint. Loud systolic bruit over heart apex. Heart's beat 170. Urine, sp. gr. 1030, acid, with a trace of albumen. He had great difficulty in swallowing. He looked about him in a wild, excited and suspicious manner; talked a great deal, but his words could not be understood.

During the first three or four days after admission he gradually improved as regards his choreic movements, but his mental condition did not change, he continued acutely maniacal, was violent, and had the delusion that his food was poisoned. Sleep was obtained each night by the administration of ʒss. of chloral. The movements entirely ceased during sleep. The treatment consisted in the giving of plenty of nutritious and easily-digested food, and a mixture of liq. arsenicalis ♏ v., tinct. digitalis ♏ x., three times a day. For the next ten days he made steady progress towards recovery, the choreic movements were much less marked, and his mental state also improved, but there still persisted irritability of temper and suspicion.

His appetite during this time was enormous, and his thirst almost unquenchable. He put on flesh at a very rapid rate, gaining no less than seventeen and a-half pounds in weight in twelve days. At the end of this time, *i.e.*, fourteen days after admission, he relapsed, all his symptoms returning in an aggravated form, and rapidly developed to a most severe degree; his movements were so strong that he had to be placed in the padded room to prevent bruising; no relief could be got by chloral and other sedatives, and he could take no food by the mouth. He became rapidly exhausted and his mind sinking into a state of semi-coma; death took place five days after his relapse, and about six weeks from the commencement of his illness. During his lucid interval he recollected everything that had taken place from the beginning of his attack.

Autopsy (eighteen hours after death). On removing the skull the brain presented a very congested appearance, all the vessels and sinuses being full. The dura mater was adherent along the longitudinal sinus. The arachnoid was not adherent to the brain tissue, but it was of a milky appearance over the ascending convolutions of both hemispheres. There was a slight excess of sub-arachnoid fluid. The circle of Willis was incomplete, there being no posterior communicating artery on left side. The only abnormal condition presentable to the naked eye found in the brain tissue was intense hyperæmia. Spinal cord normal to naked eye. Lungs airless and almost bloodless. Heart, numerous small vegetations on mitral valve, the edges of which were thickened; no other sign of endocarditis. The remaining organs were healthy.[1]

CASE II.—Clara D., æt. twenty, single, a machinist, was admitted on June 19th, 1888.

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[1] The brain and spinal cord in these cases have been examined microscopically by Dr. Handford, of Nottingham, and the result of his examination will I believe, appear concurrently with this paper.

Family history good, no taint of rheumatism, heart disease or nervous affection.

Personal History.—Was a fairly healthy girl, steady and quiet; never had any particular illness up to the present attack. Six weeks ago she was treated for irregular menstruation, and three weeks later she seemed somewhat depressed; this continued for a couple of days, when she had what her friends considered to be an hysterical fit; she lay in bed for four days, almost continuously weeping (she was menstruating at this time). Choreic movements now began to show themselves in her limbs and face, which gradually became more violent, and in two weeks she showed symptoms of insanity, had hallucinations of hearing and sight. Her movements were now so strong that it took four people to hold her in bed, and in this state she continued until she was brought to the asylum.

On admission she was in a state of continued choreic movement of all her voluntary muscles, but the spasms were not strong and were increased by any voluntary effort. Mentally she was free from excitement, and, apparently, quite conscious. It was difficult to understand what she said, owing to the spasms of the muscles of speech. She was very much exhausted. As far as could be ascertained there was no abnormal respiratory or cardiac sign, the heart's beat was however very rapid, being 140 per minute. Pulse feeble. Pupils equal and active, conjunctivæ suffused; there was nystagmus of both eyes. Temperature 102·5. Urine, sp. gr. 1020, acid, with mucous deposit, $\frac{1}{20}$ albumen, no sugar. She was menstruating. She was put on a diet of milk, eggs and beef tea, and prescribed a mixture of tinct. digitalis ♏iij, and liq. arsenicalis ♏ij, every four hours, with chloral ʒss. at bed time. During the first night after admission she had about two hours' sleep, during which the choreic movements entirely ceased; for the rest of the night she was very restless, shouted and screamed, and her movements were more violent. The next day she was better, calmer in her mind, and her spasms were much diminished, but she was very weak and exhausted. From this time to her death a week later, there were no fresh symptoms other than those of gradual and increasing exhaustion. Her choreic movements almost entirely left her, but persisted longest in the face. Her mind was fairly clear up to the last two days of her life, when she became semi-conscious, and in which state she continued up to her death.

Autopsy (nine and a-half hours after death).—On removing the skull the vessels of the dura mater were seen to be engorged, and this was the condition found in the other membranes and the brain tissue. There was a slight excess of sub-arachnoid fluid. The weight of the whole brain was 44¾ oz. The spinal cord appeared normal to the naked eye. Heart: slight thickening on edge of mitral valve, no vegetations; the other valves were healthy. All the other organs were healthy except the uterus, the cavity of which was enlarged and the mucous membrane congested, and near the os this was eroded to the extent of about half-an-inch.[1]

These cases are, I think, of sufficient rarity to merit record, and are of special interest etiologically and clinically. In the first case there was associated with the chorea, rheumatism and heart disease; in the second case there was absence of both these diseases. Mental strain was clearly an important factor in the causation in both cases; the man was very anxious about a foot-ball match he was going to play in a short time before his illness began; and the girl suffered much anxiety on account of the non-appearance of her catamenia. It is interesting clinically to note that there was a certain similarity in the mental symptoms in both cases; each had hallucinations of sight and hearing, and suspicion and irritability were marked symptoms in both.

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[1] The brain and spinal cord in these cases have been examined microscopically by Dr. Handford, of Nottingham, and the result of his examination will, I believe, appear concurrently with this paper.

ASYMMETRY OF THE OLIVARY BODIES OF THE MEDULLA OBLONGATA.

BY ARTHUR V. MEIGS,

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A LITTLE more than a year ago I was able to exhibit to the members of the Society the medulla oblongata of a baby three months of age that died of infantile atrophy, which presented a great difference in size of the olivary bodies. Examination of the sections prepared by Dr. Wm. M. Gray fails to show any abnormality in their microscopic structure, though of course the smaller olivary body presents upon its external surface a much less full and rounded curve than the other one, and the *nucleus dentatus*, is seen to be smaller and apparently less complicated in its windings. Last year I mentioned that I had seen and made sections of the medulla of a man fifty-one years of age who died of Bright's disease, whose left olivary body was much smaller than the right. In this case there was also quite marked asymmetry of the two sides of the medulla which at the time I supposed to be due to some imperfection of the methods of preservation and hardening and preparation for microscopic examination; but now I am quite satisfied of the contrary, as the same asymmetry was perceptible, though in less degree, in the case of the child above mentioned. During the last year I have had occasion to make quite a number of post-mortem examinations, and in some instances have been able to investigate the condition of the olivary bodies. Of infants I have records of five cases in which a note was made of their condition, and in two out of the five (including the one first mentioned) they were unsymmetrical. These cases were as follows—the one first mentioned, a male three months old, died of infantile atrophy (marasmus), and the left olivary was the smaller; a male infant three months old died of acute hydrocephalus, and the left olivary body was the smaller; a male infant five weeks old died of whooping cough, and the olivary bodies were symmetrical; a male six months of age died of infantile atrophy with various complications, and the olivary bodies were symmetrical; a female infant six weeks old died, apparently of kidney disease, and the olivary bodies were symmetrical.

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[1] Read before the Pathological Society of Philadelphia, February 14th, 1889.

I am sorry I have nothing more complete to record in regard to this interesting anomaly in adults, but I am almost sure that I found the condition of asymmetry present once last summer in making an autopsy at the Pennsylvania Hospital, though I have not succeeded in laying my hands upon the notes of the case. The sections of the medulla of the infant in which the anomaly was first noticed show that the left side generally is perhaps a little smaller than the right, the reduction not being confined to the olivary body alone; and this was more marked in the man who died of Bright's disease, as has already been mentioned. It is curious that in each of the three cases it should have been the *left* olivary body which was the smaller, and it is worthy of note too that they were all males. The difference in size of the two bodies is evidently due to imperfect development of the smaller one, and not to overgrowth of the larger. So far as I am aware this condition of asymmetry of the olivary bodies has not been previously noticed, though I confess that I have not made a very extensive examination of anatomical literature.

It is an interesting question whether the condition has any pathological bearing, and it has been my intention to examine carefully the medulla oblongata in cases of Bright's disease, but no sufficiently typical case has come under my notice as yet in which I was able to do so. At the present time therefore it is only possible to place my observation on record as an anatomical anomaly which is either very rare or has been overlooked.

Critical Digests.

PERIPHERAL NEURITIS.

Contribution à l'Etude des Névrites Périphériques chez les Tabétiques, par MM. A. Pitres et L. Vaillard. *Rev de Med.*, tome vi., 1886.

Des Névrites Périphériques chez les Tabèrculeux, par MM. A. Pitres et L. Vaillard. *Rev. de Med.*, tome vi., Mars, 1886.

Contribution à l'Etude de la Névrite Multiple, par X. Francotte. *Rev. de Med.*, tome vi., Mai, 1886.

Névrites Périphériques dans le Rheumatisme Chronique, par MM. A. Pitres et L. Vaillard. *Rev. de Med.*, tome vii., Juin, 1887.

Neuritis von Docent Dr. Remak, Berlin.

Neuritis fascians: Ein Beitrag zur Lehre von der Alcoholneuritis, von Prof. Dr. Hermann Eichhorst in Zurich. *Virchow's Archiv*, 112 Band, 1888.

Zur Kenntniss der acuten infectiosen multiplen Neuritis, Rosenheim. *Archiv. f. Psychiatrie, &c.*, bd. xviii.

Zur Klinik der multiplen Alcohol neuritis, von Dr. A. Witkowski. *Archiv f. Psychiatrie*, bd. xviii.

Peripheral Neuritis in Raynaud's Disease, by Joseph Wigglesworth, M.D. *Path. Soc. Transactions*, vol. xxxviii., 1887.

A Case of Multiple Neuritis, Andrew Smart, Edinburgh, 1888.

Degeneration of the Peripheral Nerves in Locomotor Ataxia, by Dr. John C. Shaw, Brooklyn, N.Y., 1888.

Peripheral Neuritis in Enteric Fever, by H. Handford, M.D. *Brain*, part xlii., 1888.

Peripheral Neuritis in Acute Rheumatism, and the relation of Muscular Atrophy to Affections of the Joints, by Judson S. Bury, M.D., 1888.

Nevrite multipla periferica recidiva. Atassia periferica, Dr. Ginho Impaccianti. *Lo Sperimentale, Sett.*, 1888.

The position of peripheral neuritis is daily becoming more and more firmly established both in the domain of pathology and of clinical medicine. But there are still many questions in both directions which urgently demand solution. And in the meantime there is rapid accumulation of cases, from which general rules will be deduced, although many of the cases, possibly, a majority, are only established clinically by conforming to *ex cathedrâ* descriptions, which a few years ago would have assigned them equally positively to anterior polio-myelites.

Pathologically the position of interstitial neuritis is clear. So too though in a less degree is that of acute parenchymatous neuritis, where there is a rapid degeneration of the myelin and the axis cylinder, with a proliferation of the nuclei of the sheath of Schwann, and the course is that followed by nerve fibres after division. But there is much doubt whether the sub-acute and chronic cases of parenchymatous neuritis (so-called) are in any degree inflammatory and are not either simple or specific *degenerations*. The latter view is gaining much ground. The neuritis of diphtheritic paralysis, from its very special features and course, would appear more than almost any other to be an example of a true neuritis due to a specific poison. And yet there are grounds for thinking it a degeneration. It can hardly be due *directly* to the organised virus—micrococcus or bacterium—which is the cause of the throat affection. The organisms would surely have been found, they would surely produce the same widespread necrosis as on the surface, and further, the patient during diphtheritic paralysis would *remain infectious*, which is not the case. That the nerve *degeneration* is due to a ptomaine produced by the growth of the organised virus in the throat and absorbed, is probable, but the supposition is not free from difficulty. And lastly, the degeneration may be simple, and not due to any poison, organised or unorganised, special to diphtheria. These three possibilities are open in several other forms of neuritis—*e.g.*, in phthisis, syphilis, enteric fever, rheumatism, &c. And the importance of settling the question is this: if the neuritis were a part of the bacterial invasion, which we believe to be the essence of the disease, we should be justified in continuing, notwithstanding our hitherto want of success, to treat the affection by remedies directed against the specific poison. But it is only a degeneration due to the cachexia left by an exhausting disease, not only would such efforts be demonstrably vain, but our course both in the direction of prophylaxis and treatment would be more plain. Rosenheim in speaking of "acute infectious multiple neuritis" says that it is not directly caused by the micro-organisms of the disease, but is the result of some poisonous product of the micro-organisms. Professor Leyden, too, on this point says: "Are we to suppose that the pathological microbes become localised in the affected nerves, and set up the neuritis? This is unlikely on several grounds. Specific micro-organisms have not yet been found in the affected nerves. Also the period at which the neuritis occurs, and the course of it, render it most unlikely that it is due to the specific micro-organisms of the infectious disease. There is no reason to suppose that the tubercle bacillus, either directly or by producing a ptomaine, is the cause of the neuritis in phthisis; rather is it a result of the general cachexia."

If this view can be maintained it greatly modifies the importance of peripheral neuritis in many diseases.

The slight implication of the sensory fibres and the remarkable variation in the sensory symptoms is a phenomenon of which many explanations have been given, but none wholly accepted. It is probable that widespread and well marked disturbances of sensation are only found when a main nerve trunk is affected; but when the disease is confined to the smaller peripheral nerves, or possibly in great measure to the peripheral end organs, sensory changes are slight or wanting. Neuritis when acute may extend along a nerve trunk in either direction by simple continuous spread of the inflammatory process. And after destruction of the conducting power of a length of nerve by neuritis, a descending secondary degeneration may extend down the nerve to the periphery. It has been shewn that sensory fibres whose peripheral portions and end organs have been removed by amputation atrophy upwards. It is extremely probable that the same is true of the motor fibres, not only after amputation, but also where the muscular fibres to which they have been distributed have long disappeared from primary atrophy or other causes. It is contrary to the well-known Wallerian law that motor or sensory fibres should *degenerate* so long as they remain in connection with their trophic centre, viz., the ganglion cells in the anterior horns of grey matter, or the ganglia on the posterior nerve roots. But it is equally contrary to general principles that fibres which are incapable of performing any function should remain for years without undergoing atrophy. This is of importance in investigating the condition of the nerves in pseudo-hypertrophic paralysis, as the atrophic nerve fibres may be mistaken for the results of a primary neuritis. The question has been raised by the present writer in a paper which will shortly appear in vol. xl. of the Pathological Society's Transactions.

In Reynaud's disease peripheral neuritis has been described by Dr. Wigglesworth but so far his observations have not yet been confirmed. The chief changes appear to have been interstitial, with some atrophy of the nerve tubules. Parenchymatous neuritis and degeneration of the nerve tubules seem to have been slight or absent, and the atrophy affected the myeline sheath rather than the axis cylinder. The case mentioned by Dr. Barlow of Reynaud's disease running on into sclerodermia —another disease supposed to depend on nerve disturbance— is worthy of notice.

It has been suggested by Mr. Jonathan Hutchinson that in circumscribed sclerodermia there is primarily a peripheral neuritis, but in no case has its existence been demonstrated yet.

In herpes there appears to be a genuine neuritis with exudation of leucocytes and other inflammatory products, and not simply a nerve degeneration. It is worthy of note too that in this disease pain is a more prominent symptom than in almost any other form even of sensory neuritis, and numbness, tingling and perverted sensations are comparatively absent.

In hemi-atrophia facialis there are grounds for believing that the nutritive changes are the consequence of a peripheral neuritis affecting especially the sensory and trophic fibres of the fifth nerve.

In tabes dorsalis, apart from pseudo-tabes or sensory neuritis, peripheral neuritis has now been extensively observed and seems to be a portion of the general degenerative process. According to Dégerine muscular atrophy in locomotor ataxy is very frequently due to peripheral neuritis and not to polio-myelitis.

Pitres and Vaillard came to the conclusion that peripheral neuritis does not play any part in the production of the specific symptoms of tabes, such as the lightning pains, inco-ordination of movement, abolition of knee jerks and disturbances of the muscular sense. These symptoms appear to be due to the sclerosis of the posterior columns of the cord, and of the posterior nerve roots. On the contrary the following symptoms which though by no means constant are not unfrequently present, seem to be directly caused by pheripheral neuritis, namely, areas of cutaneous anasthésia or analgesia; trophic cutaneous disturbances, such as perforating ulcers, skin eruptions, falling of the nails; some cases of motor paralysis, accompanied or not by muscular atrophy; the joint symptoms, and spontaneous fractures of bones. The visceral crises are also probably dependent on a neuritis of the visceral nerves.

The question has been raised by Leyden and others whether a peripheral sensory neuritis producing the symptoms of pseudo-tabes can spread to the cord and so develop a typical tabes. It is *a priori* improbable that a degeneration which spreads continuously should not be checked by the interruption in the course of the sensory fibres in the ganglion on the posterior root, but as a matter of fact the question still remains unsettled.

A case of "Relapsing Multiple Peripheral Neuritis" reported by Dr. Impaccianti is of much interest. Though called "rheumatic" the term was apparently only intended to imply that the case was supposed to be of spontaneous origin and to have followed exposure to cold and wet. The relapsing character was very remarkable and more suggestive of a malarial origin. Possibly some of the cases of malarial paraplegia reported are due to neuritis.

In chronic rheumatism (? osteo-arthritis) Pitres and Vaillard have found extensive peripheral neuritis in three cases. But they say that "it appears to result from their researches that peripheral neuritis cannot legitimately be considered as the immediate cause of the articular lesions, and of the pains which characterise chronic rheumatism; but that peripheral neuritis is met with constantly in the regions, where during life the trophic disturbances which so often complicate rheumatism are found." These are especially lesions of the skin and nails, and muscular wasting. Neuritis does not appear to play any part in the joint lesions, for MM. Pitres and Vaillard found that even in the case of the joints most profoundly altered the nerve trunks were totally unaffected. The clinical significance therefore of neuritis in chronic rheumatism would appear not to be great; in fact it seems at the best doubtful whether the neuritis is not a degeneration or atrophy secondary to the skin and muscle changes. The suggestion of the authors of a "*névrites latentes*" seems open to much objection in this disease, as in phthisis. They say:—"Dans le rheumatisme chronique comme dans la tuberculose, la fièvre typhoide, le tabes, il y a des *névrites latentes*, ou du moins des névrites *dont les symptômes échappent à l'observation*. Il n'y a pas donc lieu de sétonner que nous ayons rencontré des lésions non doubteuses dans les rameaux cutanés de certains nerfs du membre supérieur du malade qui fut l'objet de notre observation iii., *bien que l'examen clinique ne fasse mention d'aucune modification appréciable dans la structure de la peau de ces membres*."

Dr. Judson Bury has called attention to the presence of peripheral neuritis in acute rheumatism and to the relation of muscular atrophy to affections of the joints. And here again the old question arises: granting the neuritis, is it due directly to the rheumatic poison, whatever that may be, and so a toxic neuritis? This is the view of Dr. Bury, who says: "It appears to me that we have justifiable if not conclusive grounds for believing not only in a neuritis but in one set up by the rheumatic poison." This is one of the numerous questions awaiting solution. There are some strong objections to this view. The neuritis is not in any way amenable to the remedies which influence the other rheumatic processes. The ulnar nerve seems to be the one by far most frequently affected, and that often during convalescence. The ulnar nerve is specially liable to injury by pressure, especially during prostrating illnesses, and is well known to suffer during the impaired vitality and resisting power of convalescence from acute disease, and during the puerperium.

The rapidity of the muscular wasting in the neighbourhood of diseased joints was pointed out by Sir James Paget. It is to be seen also in rheumatism and is attributed by Dr. Bury to the same cause, viz.,—either a "reflex mechanism, whereby irritation conveyed along sensory nerves from the joint to the cord, inhibits in some way this functional activity of the motor cells in the anterior horns," as is suggested by the sudden onset; or as the progressive character and duration of the atrophy render more likely, "some organic changes either central or peripheral." It is very probable that in inflammatory joint affections, whether of rheumatic, traumatic, or tubercular origin, the nerves of the joints become involved in the inflammatory process, and an ascending neuritis travels speedily along them to the nearest trunk, and that the muscular wasting is thus due to a motor neuritis propagated by direct continuity. This theory is simple and will apply to all cases. It does not depend on the existence of a hypothetical rheumatic poison or on a reflex theory, the truth of which in other forms of so-called reflex paralysis there is grave reason to doubt.

The same difficulty meets us again in the neuritis of phthisis. There is absolutely no ground at present for considering the neuritis itself as a manifestation of the tubercular process, and due to the presence of the tubercle bacillus. The latter has not yet been found in the affected nerves. Another complication of this class of cases is the estimation of the influence of alcohol. In several of the instances of peripheral neuritis "*chez les tuberculeux*" narrated by MM. Pitres and Vaillard, there was also a strong alcoholic history, and in one case a history of syphilis. And how easily this complication may be overlooked, the following example, which has quite recently come under the notice of the present writer, will show. A woman was admitted into the hospital with all the symptoms of alcoholic neuritis, but she was at the same time suffering from pulmonary phthisis. Alcoholism was absolutely denied by herself and those of her friends who were seen. It was supposed possible therefore that the neuritis might be tubercular. After her death from phthisis, the relative who came for the death certificate, and who had not been seen before, said—"Ah! poor thing! she was a terrible drinker!"

The conditions common to alcoholism, phthisis, convalescence from acute disease, and the puerperium—all periods at which neuritis is specially common—are imperfect nutrition and the loading of the tissues and fluids of the body with waste products in the process of excretion. Before giving in one's adhesion to the theory of the *specific* nature of the neuritis which complicates or follows the various acute and other diseases, it is well to recall the observations of Oppenheim and Siemerling.[1] These authors found that in long-continued and exhausting diseases there is usually a very definite change in the peripheral nerves. These observations are in some measure borne out by the class of cases in tuberculous subjects, called by MM. Pitres and Vaillard, "*Névrites périphériques latentes ou ne donnant lieu à ancun trouble saillant;*" or giving rise to symptoms "*si peu accusés que leur existence échappe au médicin, et au malade lui-même.*" It is very difficult to look upon these cases of neuritis as more than a degeneration, and to assign to them any definite clinical place so long as the symptoms are so slight or even altogether absent.

The second and third of MM. Pitres and Vaillard's categories are less open to criticism and comprise those cases characterised by local muscular atrophy, or by sensory disturbances such as hyperæsthesia, anæsthesia, neuralgia, &c. But as many of these conditions are only temporary and eventually completely pass away, it makes it more and more unlikely that the neuritis is a tubercular process rather than a simple inflammation or *degeneration.* Dr. Francotte in describing a case of multiple neuritis in phthisis says, "The changes in the nerves present no sign of inflammation; it appears to be a simple degenerative atrophy," and he suggests as more exact than peripheral or multiple *neuritis,* the term *multiple atrophy of the (peripheral) nerves.*

Whether the acute ascending paralysis of Landry is due to a neuritis or not, though extremely probable, has not yet been proved. Transition cases between the very rapid progress of this disease and the slow course of most forms of pheripheral neuritis are found, and some are related by Francotte. In one case a woman, aged forty-six, free from alcoholic history, was reduced to a completely helpless condition in eighteen days, and died within twenty-six days of the onset of the nervous symptoms. In a second case a woman, aged fifty-three, was seized with pains and weakness of the right leg. A day or two later the left was affected, and two or three days afterwards the upper extremities. In *eight days* she could not move any of her limbs. In seven months she was sufficiently recovered to walk without assistance.

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[1] *Beiträge zur Pathologie der Tabes Dorsalis und der peripheren Nerven erkrankung. Arch. f. Psych.*, xviii. 2.

Rosenheim gives a very full and complete account of a man, aged thirty-five, who died in seventeen days of an ascending paralysis with signs of implication of the vagi. The illness had been preceded by an attack of intestinal catarrh for which he had received hospital treatment four months before. The fatal illness began with the feeling of the legs "having gone to sleep." In a week there was absolute loss of power, and much loss of sensibility in the legs, and considerable weakness of the upper extremities. The temperature always remained normal and there were no other prominent symptoms. At the autopsy, brain, cord and nerve roots were found perfectly normal. The most marked changes were found in the large nerve trunks, such as the sciatic, the cords of the brachial plexus, and the vagus, and consisted of hæmorrhages visible to the unaided eye, and some of them as much as one-and-a-half ctms. in length. Only a few fresh osmic acid preparations were examined, and in them the nerve fibres were healthy. In the sections made after hardening in Müller's fluid, only very indefinite changes were found beyond the hæmorrhages before mentioned, and a considerable accumulation of cells surrounding the blood vessels. The small intermuscular nerve fibres were unaltered. These appearances together with the clinical aspects of the case seemed to Dr. Rosenheim to warrant him in regarding it as one of "*acute infectious multiple neuritis*."

Professor Eichhorst has described a case of alcoholic neuritis under the title of *neuritis fascians* (*fasciare*=to enclose or tie in bundles), on account of the following peculiarities. The neuritis was solely of the usual parenchymatous type in the smaller nerve trunks, but in the smallest intermuscular nerves there was extensive interstitial neuritis; this took the unusual form of a connective tissue growth from the neurilemma enclosing some of the neighbouring muscular fibres, which consequently atrophied from pressure. Similar appearances have been described by Frankel and by Eisenlohr in the striped muscles in phthisis, and in the essential paralysis of children. Professor Eichhorst does not therefore suggest that neuritis *fascians* is peculiar to alcoholic paralysis, or that it will be found in every case of the latter.

ON THE ABSENCE OF THE CORPUS CALLOSUM IN THE HUMAN BRAIN, WITH THE DESCRIPTION OF A NEW CASE.

BY ALEXANDER BRUCE, M.D.

(Reprinted from the Proceedings of the Royal Society of Edinburgh.)

CASES of absence or defect of the corpus callosum are of interest, not only because of their great rarity, but because of the light which they throw on the distribution and functions of this commissure, and on the development of the mesial aspects of the cerebral hemispheres.

AUTHOR'S CASE.—The case here recorded came under my notice accidentally while examining the brain of a man who had died of pneumonia in the Edinburgh Royal Infirmary in October, 1886. During the short period of his stay in hospital, Dr. Sillars the resident physician noted nothing peculiar in his manner or mental condition. His sister whom I saw after his death, gave me the following account of him:—As a boy at school he was generally backward. He could read, was good at mental arithmetic, but never learned to write much more than to be able to sign his name. He was always somewhat "dour" (obstinate) and eccentric, but in no way vicious or revengeful. He was fond of music; always took an interest in what was going on around him. He was for thirteen years in the employment of one firm, where he earned a pound a week as light porter. On applying to the manager of this firm, I learned that he was considered "queer," though no one could say in exactly what way, but that he discharged his duties satisfactorily. Some time before his fatal illness he became careless and untidy in his habits, and indulged very freely in alcohol.

On removing the brain my attention was first directed to the absence of the corpus callosum. On separating the hemispheres, the frontal lobes of which were loosely united by the leptomeninges, it was seen that this commissure was completely absent, as was also the psalterium of the fornix. Covering the third ventricle and the sides of the optic thalami was a thin membrane (evidently the velum interpositum), extending from the lamina terminalis in front backwards over the thalami, and having in the middle line two long antero-posterior veins. This structure had extended into the lateral ventricles, and was fringed by the choroid plexus in the usual way. It was loosely connected with the falx, but the adhesions were torn in removing the latter. The two hemispheres were separted by a mesial incision and placed in Müller's fluid; the left reserved for transverse vertical, the right for transverse longitudinal sections. Nothing abnormal was noted about the size or conformation of the cranium, but unfortunately no careful examination of this was made. The brain was not weighed, but its size seemed fairly normal. It was richly convoluted, but there was a remarkable anomaly in the formation of the various lobes (see figs. 1 and 2—drawings natural size of inner and outer surface of right hemisphere).

The outer surface of the cerebrum presented the following abnormalities:—(*a*) The frontal lobe is reduced in size, while the occipital and to a less degree the temporal are increased. The length of the convex margin of the great longitudinal fissure between the extreme point of the occipital and frontal lobes is 11½ inches; the distance between the tip of the frontal lobe and the superior extremity of the fissure of Rolando (*f.r.*) is 3¾ inches; that between the fissure of Rolando and the parieto-occipital (*p.o*) fissure is 4 inches; and that between the parieto-occipital fissure and the tip of the occipital lobe is 3⅛ inches. (*b*) Both limbs of the fissure of Sylvius (*f.s.*) are normal, but the fissure of Rolando (*f.r.*), instead of having the normal direction downwards and forwards, passes downwards and slightly backwards. It also reaches the median surface of the hemisphere, where it extends as a deep fissure as far as the free margin of the grey matter of the gyrus fornicatus.

In the frontal lobe the sulci are all present, but the convolutions, especially the lower, are abnormally small. The præcentral sulcus (*pr.c*) and ascending frontal convolution (*a.f.*) are normal.

The postcentral sulcus (*po.c*) extends from ⅛ inch above the horizontal limb of the fissure of Sylvius to within 1 inch of the middle line. It is not directly continuous with the intra-parietal sulcus (*i.p.*) which is unusually deep and extends backwards to within an inch of the parieto-occipital fissure. The convolutions of the occipital lobes are unusually large and numerous. In the temporal lobe the sulci are normal, and the convolutions (t_1, t_2, t_3) well developed.

On the median surface (fig. 2) the calloso-marginal fissure cannot be traced. The fissure of Rolando (*r.f.*), as already stated extends as a deep vertical cleft almost to the free edge of the grey matter. The parieto-occipital (*p.o*) and calcarine (*c*) fissures, both of which are well marked, do not join each other, but each passes separately into the fissura hippocampi. The parieto-occipital fissure is unusually far forward so that on its mesial aspect also the occipital lobe is unusually large.

On this aspect of the frontal lobe are several quite anomalous fissures. Their distribution is very accurately represented in the drawing (fig. 2). Specially noteworthy are two almost horizontal sulci (*f.h.*) joining the anterior upper angle to the triangular area *spt.* These probably represent the anterior end of the embryonic fissura hippocampi (fig. 31; *cf.* also figs. 11, 12, 16, 21). On the parietal lobe, between the (anomalous) fissure of Rolando and the parieto-occipital fissure (*po*), are two deep sulci which pass at a distance of about ½ inch from each other from the free lower margin of the gyrus fornicatus almost to the vertex. They lie near the middle of the lobe and diverge slightly from each other as they pass outwards. In consequence of the absence of the calloso marginal sulcus, and of the peculiar distribution of the other fissures, the gyrus fornicatus is apparently gone, and the convolutions on this surface have a peculiar radiated arrangement (*cf.* figs. 12, 16, 21, and see Case X.).

The hippocampal (*h*) and the uncinate (*u*) gyri are normal.

The convolutions on the inferior aspect followed the normal type.

On the base of the brain, the vessels, optic nerves (*o.n.*), chiasma (*o.c.*), and tracts were normal, as were also the corpora albicantia (*c.m.*) and the peduncles.

On the mesial aspect the following structures were present and normal (see fig. 2):—(1) the anterior (*a.c*), middle (*m.c.*), and posterior commissures (*p.c.*); (2) the optic thalamus and infundibulum; (3) the lamina terminalis (*l.t.*).

The corpus callosum was entirely absent. The septum lucidum and fornix were apparently absent; but, placed more laterally than these structures, and overhung by the grey matter of the cortex, a triangular area of white matter (which has the size represented in the drawing—*spt.* fig. 2) lay between the anterior commissure below, the free edge of the grey matter (of the gyrus fornicatus?) in front and above, and the tela choroidea (not shown) and optic thalamus behind and below. This area has several shallow longitudinal grooves. Its lower rounded margin is formed by a structure, which is undoubtedly the fornix (ascending limb). This triangular area is almost certainly the septum lucidum (see below).

Transverse vertical section of left hemisphere (fig. 3) made immediately anterior to the triangular area of white matter (*spt.* fig. 2), and through the anterior cornu of the lateral ventricle. *c.n,* caudate nucleus; *l.n,* lenticular nucleus; *i.c,* internal capsule (*of quite normal, size and appearance*); *c.r,* fibres of corona radiata curving from internal capsule upwards and mostly inwards towards grey matter of convolutions, almost no fibres traceable into the dark area *spt*; *spt,* a dark area of fibres having mostly antero-posterior direction regarded as a forward continuation of fibres of *spt.* (fig. 2), and as belonging to septum lucidum; *l.v,* anterior cornu of lateral ventricle; *f,* between *l.v.* and *spt,* white fibres running upwards and outwards, and then entering tract *spt.*; and *possibly* belonging to fornix system (a similar strand seen in brain of kangaroo—Beevor): *e.c,* external capsule; *cl,* claustrum; *f.s,* fissure of Sylvius.

Fig. 4. Transverse section at level of anterior commissure. *a.c,* anterior commissure (of normal size); *f,* fornix, ascending limb (relation to *spt.* should be noted); *spt,* an area of white fibres—mostly having a longitudinal direction—a few strands crossing it transversely cannot (microscopically) be traced further than a dense network at its outer edge; *c.s,* a shallow fissure between *spt.* and gyrus fornicatus (*g.f.*), regarded as representing the callosal sulcus; *i.c,* internal capsule—careful examination shows to be quite normal size; *c.r,* coronal radiata—passing upwards and inwards. Many fibres traced into (*g.f.*), gyrus fornicatus. A few seemed to enter the network outside area *spt.*

Fig. 5. Transverse section, made at posterior limit of the triangular area *spt.* (fig. 2), and about the middle of optic thalamus; *r.b,* an oval area of white fibres, mostly running longitudinally; several strands run transversely into the irregular network on its outer margin; this network passes round lateral ventricle within the internal capsule and may be connected with (*c.n.*) caudate nucleus; the strand *r.b.* is evidently the backward prolongation of strand *spt.*; *f,* fornix—of normal size, but very lateral in position, intimately connected with the strand *r.b.*; *g.f,* gyrus fornicatus; *c.s,* callosal (?) sulcus—between *g.f.* and *r.b*; *i.c,* internal capsule—again normal in size; *c.r,* corona radiata—many fibres again traced over the area *r.b.* into gyrus fornicatus, as well as into other convolutions at vertex; *o.t,* optic

thalamus; *e.c*, external capsule; *cl*, claustrum; *i*, island of Reil; *t*, temporal lobe; *f.s*, fissure of Sylvius; *o*, optic tract.

Fig. 6. Transverse section through pulvinar of optic thalamus; *r.b.*, backward continuation of area *r.b.* (fig. 5), some of its fibres traced outwards for a short distance (see the dark shaded part) along upper wall of lateral ventricle; *f.*, fornix, body, in intimate relation to area *r.b.*; *f.*, fimbria of fornix, in intimate relation to (*g.d.*) fascia dentata, and (*c.amm.*) cornu ammonis.

Fig. 7. *l.v.*, posterior cornu of lateral ventricle, much dilated; *o.r.*, optic radiation of Gratiolet (*cf.* figs. 20 and 25); *t.*, a thin band of fibres, between optic radiation of Gratiolet and ependyma of ventricle. Note the absence of all callosal fibres. This tract has been very carefully drawn from both naked eye and microscopic sections. *i.l.f.*, inferior longitudinal fasciculus.

Fig. 8. Transverse longitudinal section of right hemisphere above the level of the lateral ventricle. Shows the remarkable shortness of the frontal lobe; *f.r.*, fissure of Rolando; *f.r.x*, the abnormal Rolando (fig. 2) on the mesial aspect of the hemisphere. The crowded grouping of convolutions at the bottom of the fissure should be noted. This probably explains the shortness of the frontal lobe, the gyri, which should normally have been on the mesial surface, and extended round the tip of the lobe, being compressed into this position. In the absence of evidence of constriction by any malformation of the falx or membranes, it is probably a result of repression of the forward growth of the hemisphere during its development.

Fig. 9. Transverse longitudinal section of same hemisphere above the level of the optic thalamus (seen from below, to show the arched form of the structures *spt* and *r.b.*). *f*, fornix.

Fig. 10. Similar section slightly lower than the fig. 9 (from above). Letters as in preceding sections. Note *spt.* as a broad strand of white fibres lying internal to the anterior horn of the ventricle (represented by a black line). Its fibres pass from below, backwards and upwards, and enter *r.b.* (fig. 9). Note that in fig. 9 *r.b.* is arched, and has the fornix along its inferior surface. *o.r.*, optic radiation of Gratiolet; *t.*, a narrow strand (drawn exactly of natural size) internal to *o.r.*, and representing the tapetum, which remains when the forceps major is removed (note absence of all callosal fibres). The disproportion in size between the structures marked *t.* and *spt.* is to be noted (see cases of Onufrowicz and Kaufmann). In fig. 10 the apparently normal relation of fimbria of fornix (*f.x.*), fascia dentata (*f.d.*), and cornu ammonis (*c. amm*) is to be noted. In the section from

which fig. 9 was drawn the mass of the fibres of $r.b.$ passed into the white investment of the cornu ammonis.

Apart from the absence of the great transverse commissure, the points of special interest in the above case are the deformity of the frontal lobe, the peculiar radiated arrangement of the convolutions on the median aspect of the hemisphere, the value of the structures *spt.* and *r.b.*; the relation of the callosal fibres to the internal capsule (with reference to Hamilton's recently expressed views), and finally the light thrown on the ordinarily accepted opinions with regard to the functions of the corpus callosum.

With a view to their elucidation I have abstracted the accounts of all the recorded cases available to me. The most important papers are in the *Archiv für Psychiatrie*, vol. i. (Sander), vol. xviii. (Onufrowicz and Kaufmann), and in the *Glasgow Medical Journal*, 1875 (Knox). It is much to be regretted that the accounts are in most cases extremely meagre and evidently frequently inaccurate.

1. *Cerebrum Divided into Two Hemispheres, but Corpus Callosum completely absent.*

I. Reil, *Arch. f. Physiologie*, xi., 1812, p. 341, quoted by Sander, *Arch. f. Psychiatrie*, vol. i. p. 135.—Woman, aged thirty; stupid, could go messages; otherwise healthy; died suddenly from an apoplectic seizure. Ventricles moderately distended with fluid. Corpus callosum completely absent. Hemispheres held together only by anterior commissure, optic chiasma, isthmus of crura cerebri, and corpora quadrigemina. Inner surfaces of anterior lobes of hemispheres completely separated; parts of them in which the beak and knee of the corpus callosum should have been inserted covered with convolutions. Fornix arose from thalamus, formed corpora mammillaria, ascended behind anterior commissure, coalesced on both sides with that part of the roof of the cerebral ventricles which runs just under the longitudinal convolutions, and formed with it a rounded edge. It ended in a normal manner posteriorly.

II. Ward, *London Medical Gazette*, March 27, 1846; see Knox, *Glasgow Medical Journal*, April, 1875.—An illegitimate child; died at eleven months; could see and hear; gave no indication of intelligence; cried like a puppy. Skull twice normal thickness. No trace of corpus callosum, anterior, middle, or posterior commissures (of fornix and septum lucidum, no note). Frontal lobes flattened.

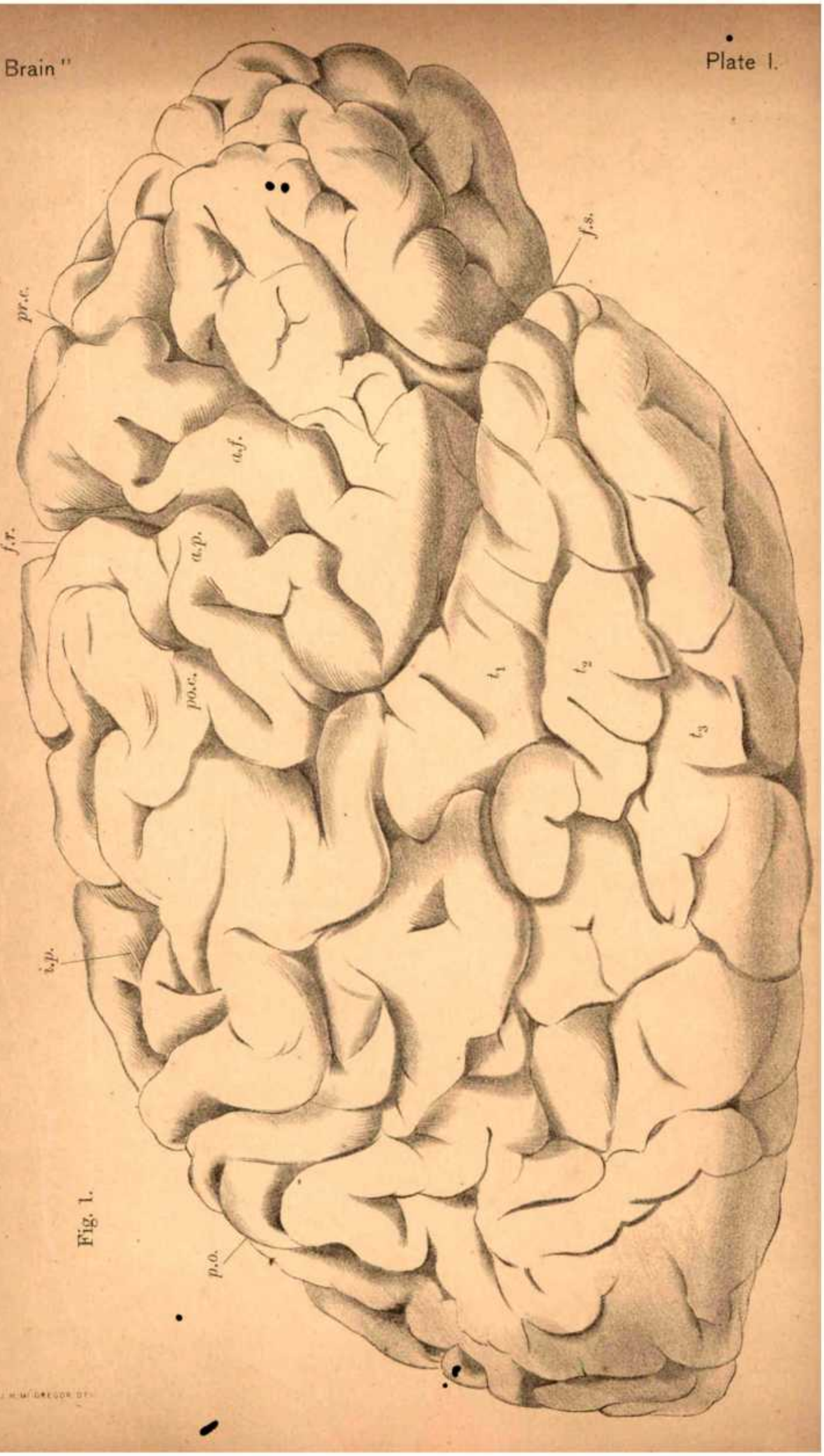

Fig. 1.

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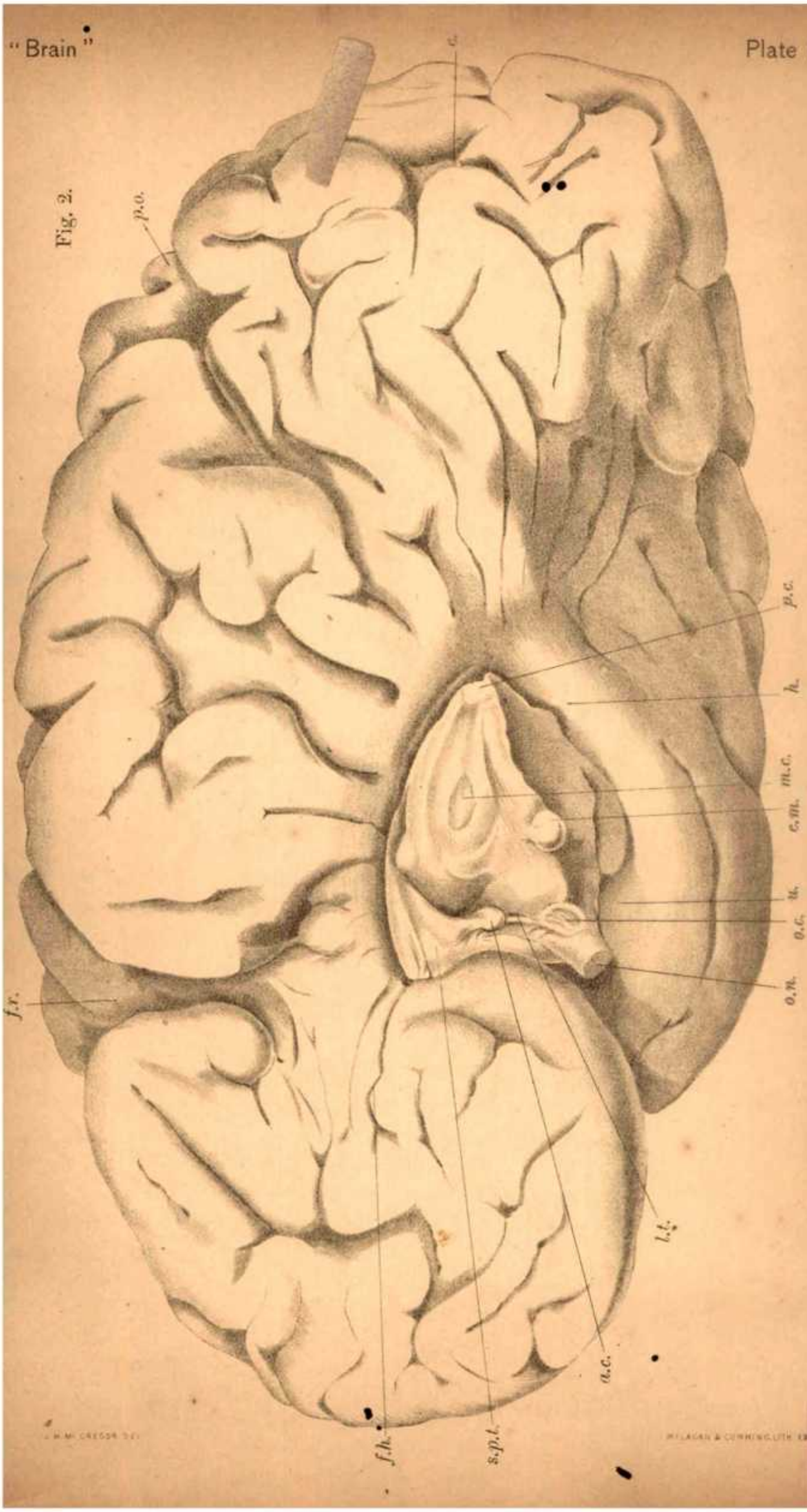
"Brain"

Plate II.

Fig. 2.



J. H. McGregor del.

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Fig. 3.

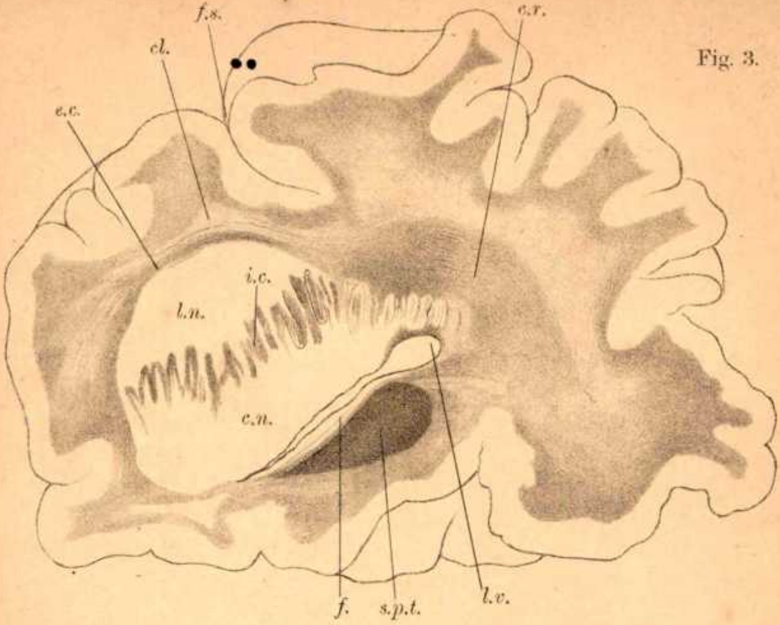

Fig. 4.

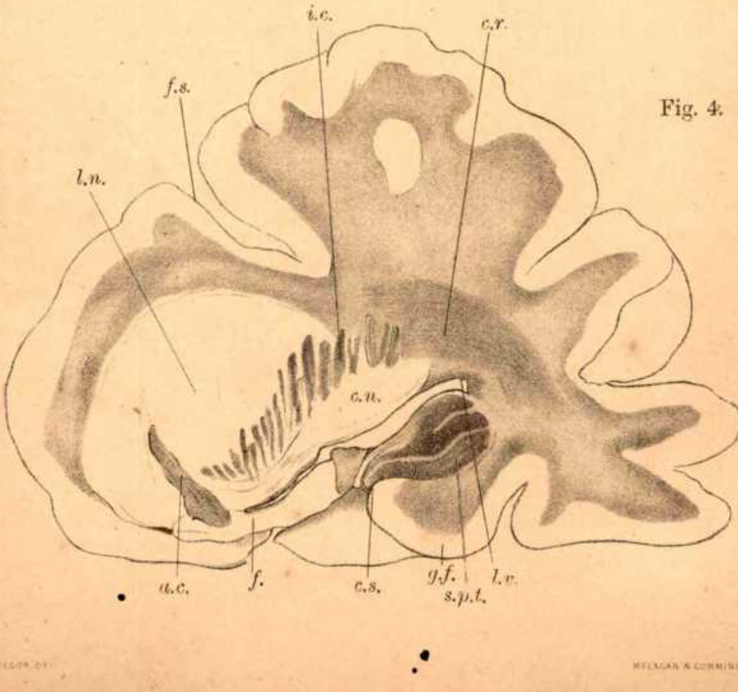

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Plate IV.

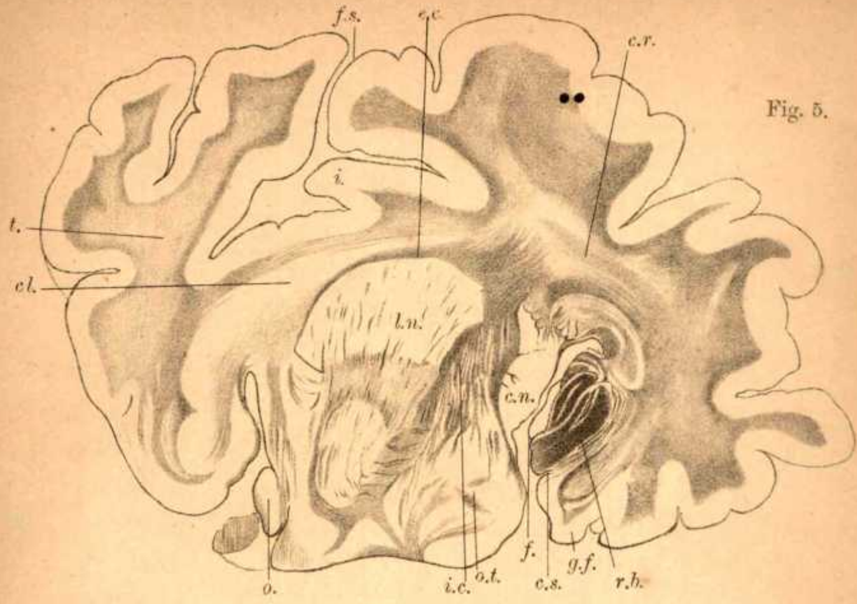

Fig. 5.

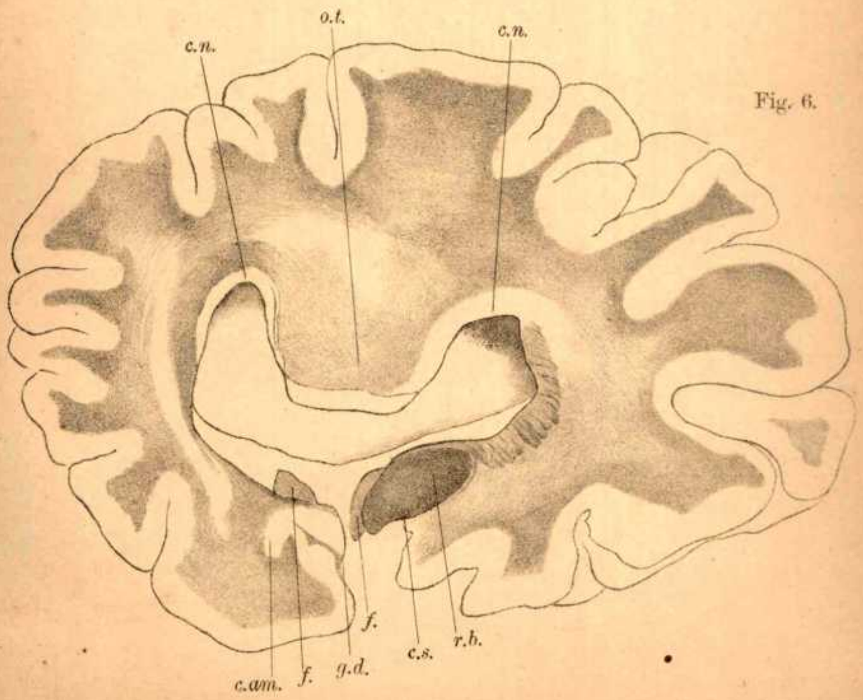

Fig. 6.

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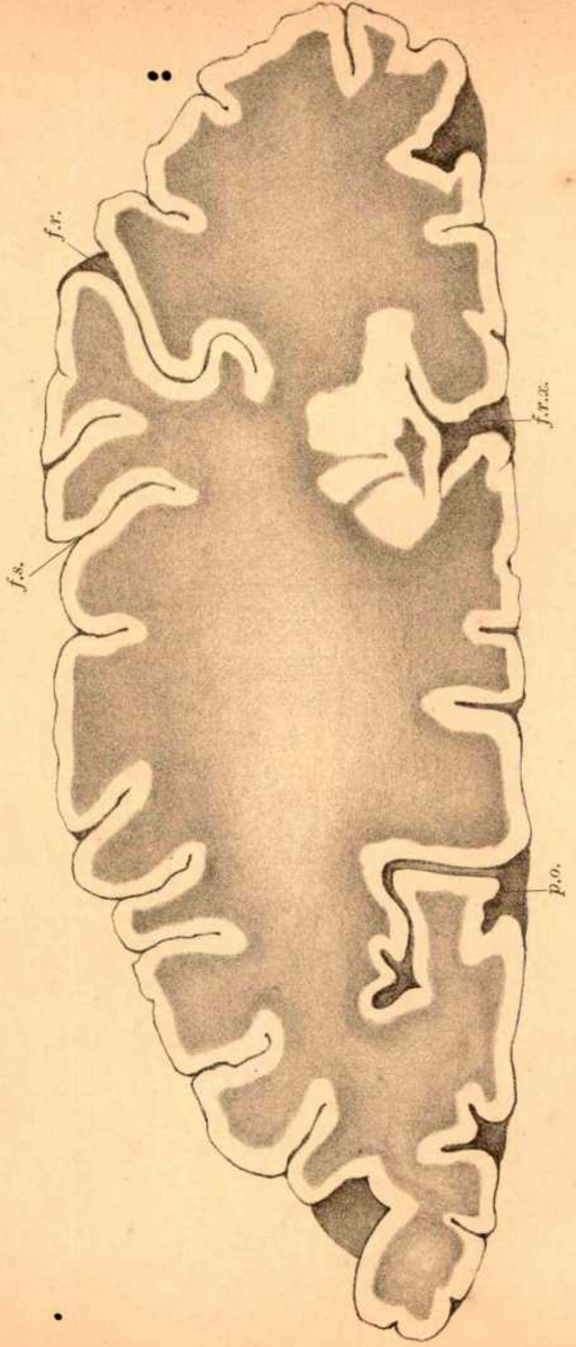
"Brain"

Plate V.

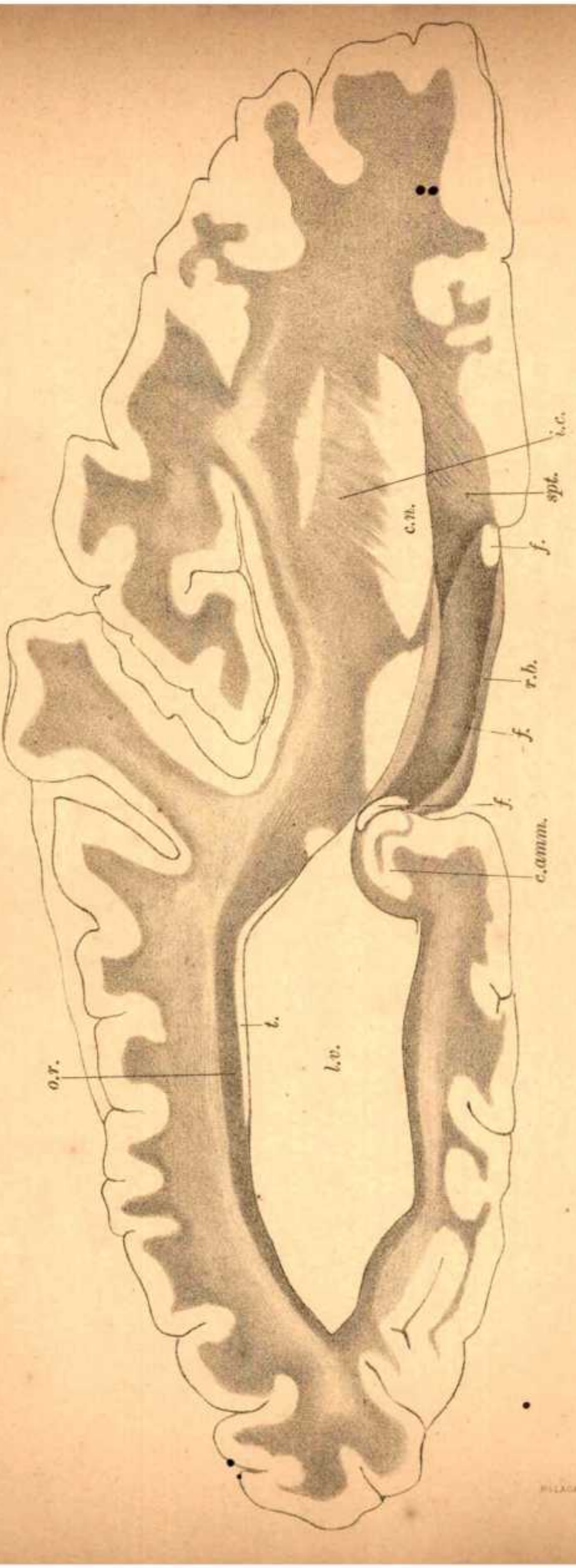
Fig. 8.



J. M. Mc GREGOR, DEL.

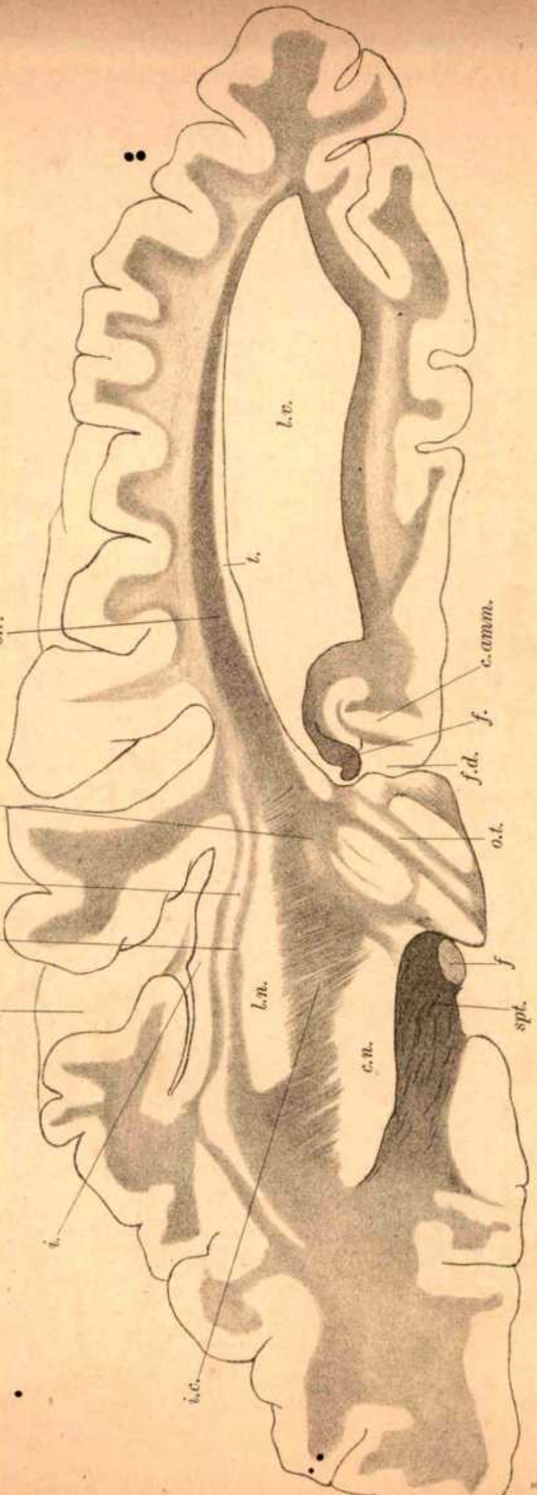
McLAGAN & CUMMING LITH. EDINR.

Fig. 9.



J. W. McGregor, del.   M'Lagan & Cumming, Lith. Edin.

Fig. 10.



J. H. McGregor, Del.

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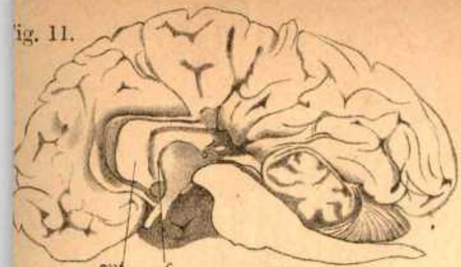
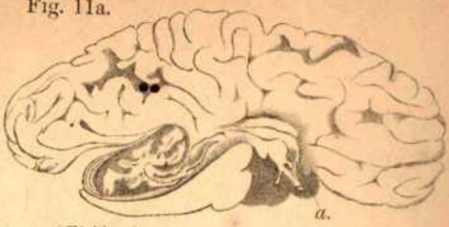
Plate VIII

ig. 11.



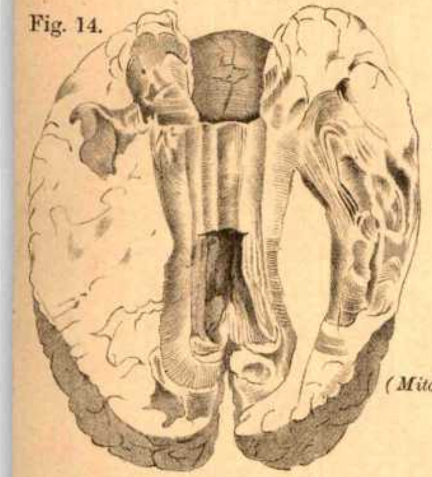
(Eichler.)

Fig. 11a.



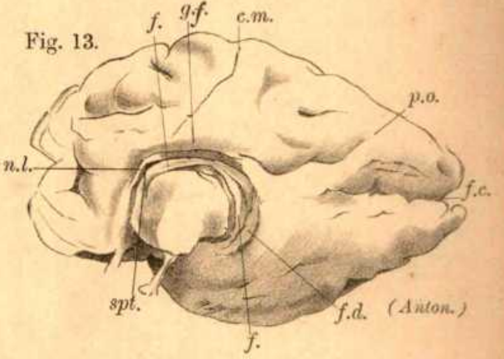
(Eichler.)

Fig. 14.



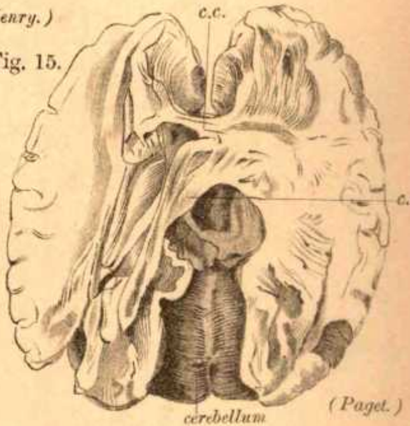
(Mitchell Henry.)

Fig. 13.



(Anton.)

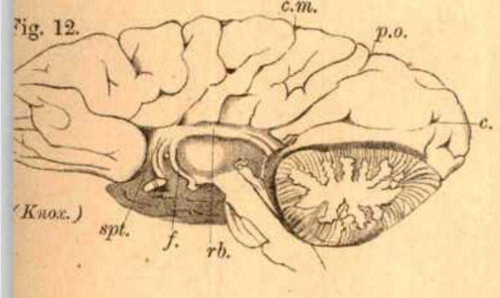
Fig. 15.



cerebellum

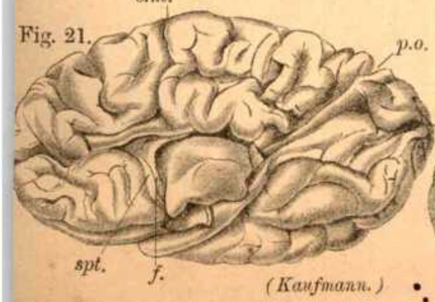
(Paget.)

ig. 12.



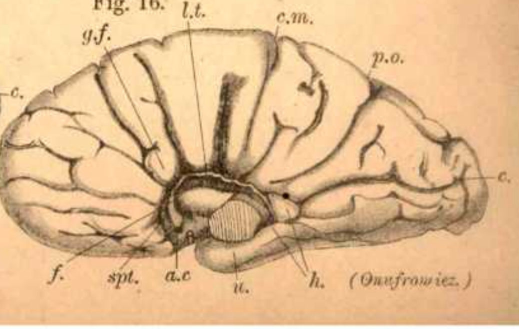
(Knox.)

Fig. 21.



(Kaufmann.)

Fig. 16.



(Onufrowicz.)

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Fig. 17.

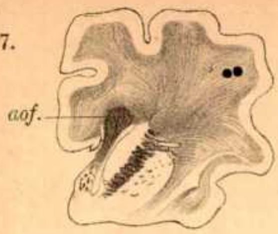

Fig. 18.

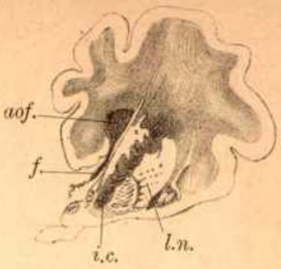

Fig. 19.

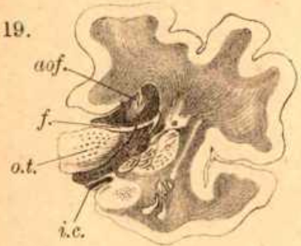

Fig. 20.

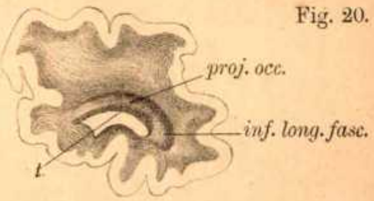

Fig. 22.

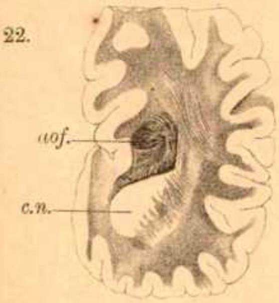

Fig. 23.

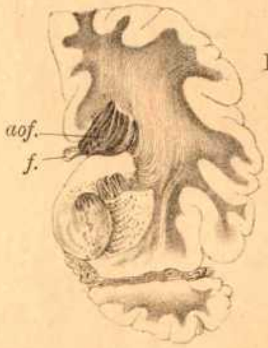

Fig. 24.

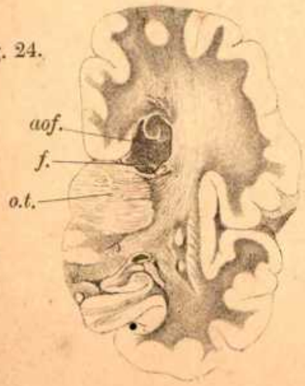

Fig. 25.

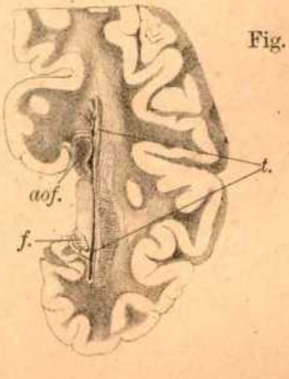

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Plate X.

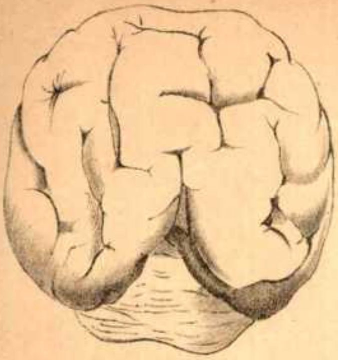

Fig. 26. *(Hadlich.)*

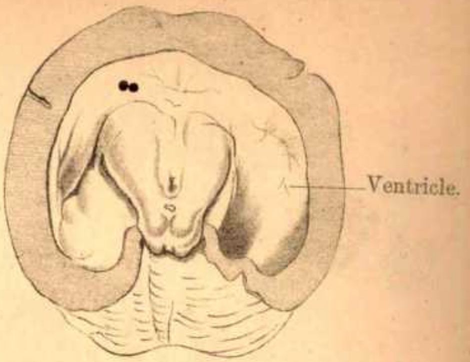

Fig. 26a. *(Hadlich.)*

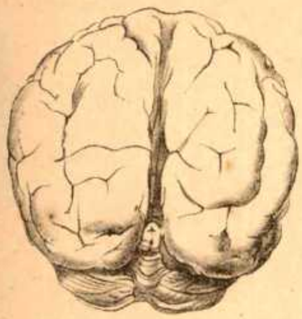

Fig. 27. *(Hadlich.)*

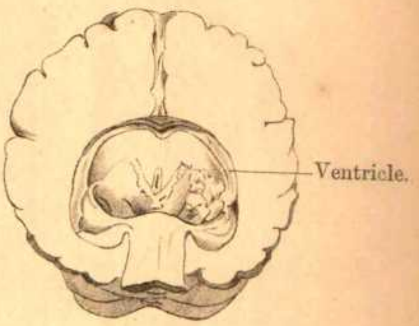

Fig. 27a. *(Hadlich.)*

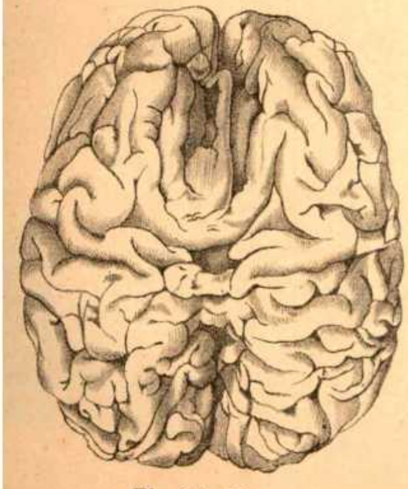

Fig. 28. *(Turner.)*

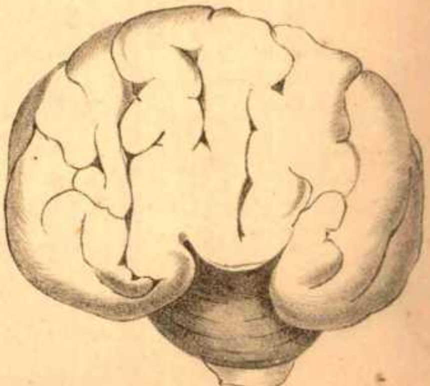

Fig. 28a. *(Wille.)*

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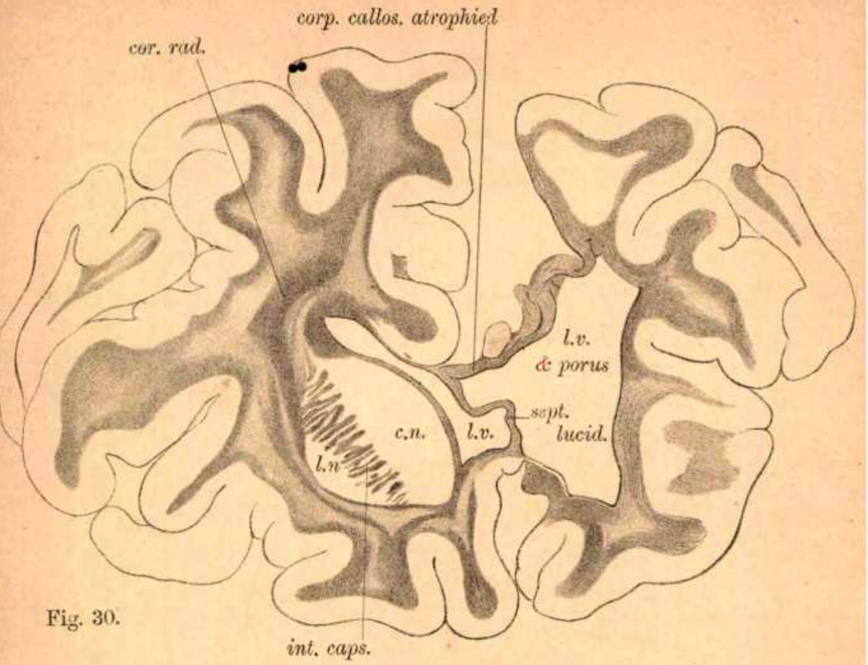

Fig. 30.

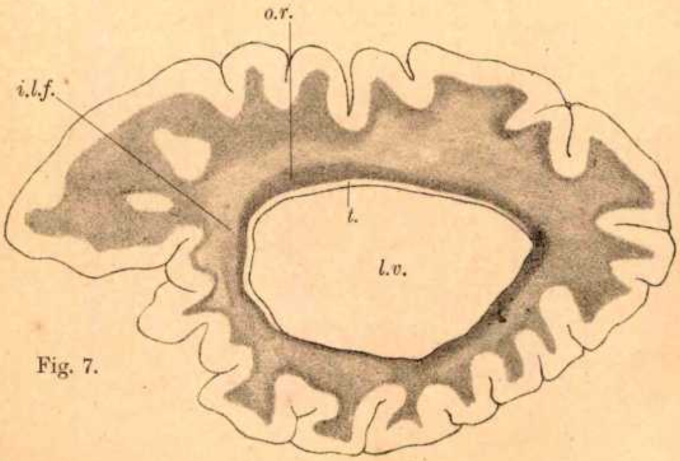

Fig. 7.

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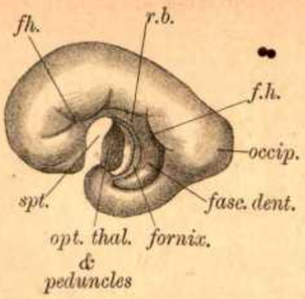
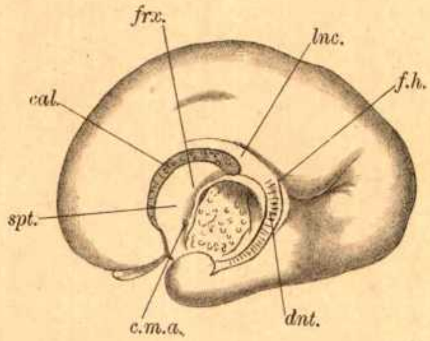
Fig. 31.



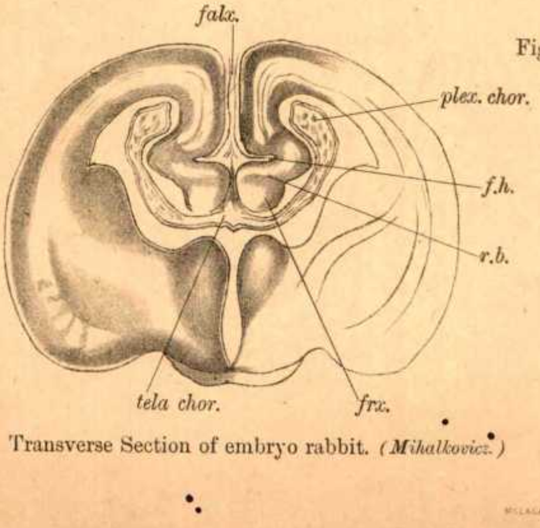
Embryo 3½ months *(Mihalkovicz.)*

Fig. 32.



Embryo 4½ months. *(Mihalkovicz.)*

Fig. 33.



Transverse Section of embryo rabbit. *(Mihalkovicz.)*

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III. *Aertzliche Berichte der Wiener Irrenanstalt für* 1853, Wien, 1858, p. 189; see Sander, *loc. cit.*, p. 135.—Male, twenty-five years, since twenty epileptic owing to fright; ultimately imbecile. Corpus callosum entirely absent. Lateral ventricles, especially in posterior horns, much dilated. Fornix seems to have been normal (no note about commissure of the body). Anterior commissure was "ein dunner beiderseits abgerundeter, sich in ein gegenüber-stehenden Stumpf endigender Balken." Nothing said about commissura mollis.

IV. Foerg, *die Bedeutung des Balkens im menschlichen Gehirn*, München, 1855; Sander, *loc. cit.*, p. 135.—Girl, aged seventeen; extremely idiotic, muscular development very feeble. Corpus callosum absent. Psalterium of fornix absent, fornix otherwise normal. Fibres of cingulum (Zwinge) on both sides [illegible]ted with fornix. Presence of anterior commissure doubtful; middle commissure absent. Lateral ventricles dilated.

V. Poterin-Dumontel, *Gaz. Med. de Paris*, No. 2, 1863, pp. 36-38; see Sander, *loc. cit.*, p. 135.—Man of seventy-two years. During the twenty-five years that he was under observation he had three or four apparently slight epileptic attacks (éblouissements passagères avec pâleur de la face et résolution momentanée des membres). Very weak-minded, but could answer simple questions correctly, and could go messages. Could read and write. Moderate œdema of lepto-meninges. No trace of corpus callosum. Lateral ventricles greatly dilated (this attributed to absence of [illegible] callosum). Commissure anterior and mollis present, [illegible] fornix (its psalterium absent). Brain weighed 1,078 [illegible] Two hemispheres were slightly asymmetrical.

VI. Huppert, *Archiv* [illegible]*de*, 187[illegible] by Knox.—An epileptic [illegible] 1,270 grammes. D[illegible]

Gazette Med. de Paris, January 16, 1875; see Knox, *l.c.*—Soldier, aged forty; of ordinary intelligence, but with a slight tendency to melancholia and taciturnity, and untidiness in his habits. Brain shows absence of corpus callosum, septum lucidum and gyrus fornicatus. (The latter two statements must be received with caution.)

VIII. Knox, *Glasgow Med. Jour.*, April 1, 1875, p. 227.—Female, aged forty; extremely idiotic, muscular system well developed. (For further details see original article.) Head of normal size, forehead low, occiput flat, brain 36½ ounces; hemispheres nearly symmetrical. Posterior horns of ventricles dilated, ependyma thickened (fig. 12). "The corpus callosum appeared to be wholly awanting, or only represented by a very slight ridge (*rb*), which anteriorly was scarcely perceptible, but posteriorly was about one-tenth of an inch in depth. It began in front above the lamina cinerea and passed upwards and backwards attached to the side of the general cavity of the ventricle, forming the upper border of a layer of white matter, the lower border of which was part of the fornix. About half-way back it became separated from the fornix, and at last ran into the anterior and lower part of the hippocampal convolution. The lamina cinerea was divided superiorly so as to appear like a small ridge running up in front of the anterior commissure. The fornix (*f*) was completely divided in the middle line. Its anterior pillars could be [illegible] to the corpora albicantia. Each lateral half ran upwards [illegible] kwards as a sharp well-defined border and might be [illegible] o the descending cornu of the lateral ventricle where it [illegible] al manner. Extending between the anterior part [illegible] ribed [illegible] s corpus callosum, was a [illegible] le thickness, apparently [illegible] but bounding on the [illegible] dently the margin of the ventricle. This anomaly is ascribed to the absence of the gyrus fornicatus. (For convolutions and sulci on outer surface, see original. They presented slight abnormalities).

IX. Eichler, *Arch. f. Psychiatrie*, vol. viii. pt. 2, 1878, p. 355.—Labourer, forty-three, married; father of well-developed child; died of gangrene of scrotum. No mental peculiarity; a diligent, capable workman; good husband in every respect; sober, quiet, well behaved; could read and write. Cerebral hemispheres asymmetrical. Brain otherwise well developed, richly but irregularly convoluted. Gyrus fornicatus absent, or indistinguishable. Calloso-marginal, parieto-occipital, and calcarine fissures indistinguishable. No corpus callosum; in its place a thin transparent membrane with some vessels on its upper surface (the tela choroidea superior?). This was probably adherent to falx, and ruptured on removal of the latter. Of the commissures, the anterior (*a*) was present and enlarged; the posterior present, of normal size; the middle absent. Fornix present, its psalterium
abse septum lucidum (*spt*) probably present, as Eichler's
fig l and 11*a* represent two laminæ in the position of the
t ar area (*spt*) in my case. Lateral ventricles dilated in
sterior horns (because corpus callosum absent.) Lepto-
s normal. On the medial surface the pia continued
rds to the margin of the fornix; the choroid plexus
the proper covering of the third ventricle absent, pro-
m in removing the falx of the dura. Lamina terminalis

rquhart, *Brain*, Oct. 1880.—Female, idiot, with deficiency
linating power over muscles. Attention, imitation, idea-
moral sense feebly developed. Calvarium thin, ex-
irregular in shape, shortened antero-posteriorly, nearly
Right side of skull flattened posteriorly, bulged slightly
ly, so that the hemisphere of that side was, as it were,
forward. Dura mater non-adherent. Cerebral hemi-
small. Convolutions small and simple, especially in the
and occipital lobes. Corpus callosum represented by
mentary ridge on each hemisphere. (From a drawing of
in kindly sent me by Dr. Urquhart, I take this to closely
le the ridge at the upper part of the white septum lucidum
own case.) Gyrus fornicatus absent, numerous radiating
co lutions taking its place. Fornix and septum lucidum absent. A thin pellucid extension of pia mater seemed to connect the hemispheres.

XI. Anton, *Zeitschrift f. Heilkunde*, vii. Bd. i. pp. 53-64, 1886 (fig. 13).—Fœtus, female. Born at seventh month; lived six hours. Skull normal in size and configuration. Falx major normal. Lepto-meninges not thickened. Both hemispheres nearly symmetrical. Poorly convoluted. Corpus callosum and psalterium of fornix quite absent. Anterior commissure also absent. Only trace of septum lucidum (*spt*) present. Fornix system (*f*) well developed; the lepto-meninges came into direct contact with it. Middle commissure of normal size. Gyrus fornicatus (*g.f.*) small. Calloso-marginal sulcus (*c.m.*) only present in its posterior vertical part. Nervus lancisi (*n.l.*) fused with the fornix, and passes into the fascia dentata (*f.d.*). Parieto-occipital (*p.o.*) and calcarine (*f.c.*) fissures do not unite. The lateral ventricles so dilated that Anton considers hydrocephalus to be the cause of the condition, and to have acted before the fourth month.

XII. Onufrowicz, *Arch. f. Psychiatrie*, xviii., 1887, p. 306, figs. 16, 17, 18, 19, 20.—Male, aged thirty-five. Died of pneumonia; extremely idiotic. (The very full description in the original article should be read.) Brain very small; convolutions on median surface show the apparent absence of the gyrus fornicatus (fig. 16, *g.f.*); the calloso-marginal fissure (*c.m.*) present only in its posterior part; parieto-occipital (*p.o.*) and calcarine (*c*) fissures do not meet; gyrus hippocampi (*h*) and gyrus uncinatus (*u*) well developed. (There are other abnormalities not of special interest here.) Corpus callosum absent; in its place a thin membrane (*l.t.*), which must be considered as the representative of the lamina terminalis (the tela choroidea superior). Psalterium of fornix absent; fornix (*f*) and septum lucidum (*spt*) displaced laterally. Anterior commissure (*a.c.*) present; middle absent; posterior cornu of latter ventricle (*l.v.*) dilated. On transverse sections a structure (see figs. 17, 18, 19, 20, *aof*) similar to that marked *spt* and *rb* in my case, and lying between grey matter and fornix, and considered to pass backwards into tapetum (*t*, fig. 20). Onufrowicz considers this strand the fronto-occipital association bundle, rendered prominent owing to the absence of the corpus callosum.

XIII. Kaufmann, *Arch. f. Psych.*, xviii. and xix. p. 769, figs. 21, 22, 23, 24, 25.—Female, twenty-four. After an accident at four years of age, her mental development was retarded and her general health impaired. When in hospital she showed feeble mental capacity without any very marked psychical change. Died of chronic parenchymatous nephritis. Skull symmetrical. Dura mater normal; pia mater œdematous, and slightly th

tal lobes with included dura and pia united together.
llosum absent; in its place a thin fold of pia mater
s in front with that lying between the two frontal lobes.
ra media absent; commissura anterior and fornix (*f*)
horoid plexus and lateral ventricle present. The fornix
the inferior margin of a strand of white fibres (*aof*),
ostly in an antero-posterior direction. This is con-
the association system of frontal and occipital lobes,
rior longitudinal fasciculus of Burdach); which has be-
minent owing to the absence of the corpus callosum
v of Onufrowicz). Gyrus fornicatus absent or rolled
owards the association bundle, but *separated from it by
issure.* Calloso-marginal sulcus (*c.m.*) abnormally far
(?). Parieto-occipital (*p.o.*) and calcarine (*c*) fissures do
. A series of transverse sections are figured (see figs. 22,
5), showing the relation of the so-called occipito frontal
tion system to the fornix and gyrus fornicatus, and to the
um of the posterior cornu of the lateral ventricle. He
tes from Wernicke (Lehrbuch) to show that this system
sses in the substance of the white fibres of the gyrus fornicatus
long its whole length round the splenium of the corpus callosum
into the gyrus uncinatus. Here, *loc. cit.* p. 231, he traces this bundle outwards over the lateral ventricle into the tapetum. How it gets back from there to the gyrus uncinatus is not very easy to understand. The cause of the lesion is supposed to be early hydrocephalus.

XIV. Christie, *Proceedings of Roy. Med. Chir. Soc.*, 1868, ref. in *Lancet*, 1868, p. 436.—Male; aged twenty; idiotic, and without power of speech from birth. Brain weight, 28½ oz.; corpus callosum completely absent.

XV. A. Virchow; Berlin, *Gesellschaft f. Psychiatrie und Nerven.*, 9th May, 1887, quoted by Kaufmann, *loc. cit.*, p. 236.—Child died at six weeks with convulsions. Marked hydrocephalus; no corpus callosum, no anterior commissure, no septum lucidum (no note of fornix). Many other developmental defects, and changes of inflammatory origin, such as thickening of pia, and adhesion to brain substance.

2. *Primary Partial Development of Corpus Callosum.*

XVI. Sander, *loc. cit.*, p. 128; *Archiv f. Psychiatrie*, vol. i., p. 128.—Cretin, brain abnormally small, corpus callosum present, but splenium reduced to ¼ centimetre, while genu is ⅝ centimetre

in thickness; psalterium of fornix present; fornix, pe
campi, calcar avis, normal; posterior of cornu of vent
normally wide, forceps of corpus callosum quite absent.

XVII. Sander, *loc. cit.*, p. 299.—Microcephalic
months old. Corpus callosum present, but splenium
forceps present; anterior commissure present, middle co
absent; fornix present, small; septum lucidum norma
ventricle not dilated.

XVIII. Sander, *loc. cit.*, p. 303.—Microcephalic brain
callosum short, splenium thin; no further examination a

XIX. Paget, *Med. Chir. Trans.*, 1846, p. 55, fig. 1
twenty-one; mental condition fairly normal; showed
want of forethought, some flightiness of manner, but had
memory, was trusty and competent, and of good ch
Convolutions normal; corpus callosum 1·4 inch long,
margin 1·9 inch from tip of frontal lobe, posterior 3·7
from occipital lobe; length 1 inch in middle line, increa
length as it proceeds outwardly. Fibres of anterior part
tinued into frontal lobes fibres of middle part—a few fibres p
transversely from one hemisphere to another; most pass wit
varying degrees of obliquity, most of the oblique bands pass from
left to right—these in the left side being thicker. There is not;
in their usual position, a trace of the septum lucidum or middle
part of the fornix. The tapetum present, psalterium of fornix
absent; fornix, anterior and posterior commissure normal, middle
commissure very large (fig. 15).

XX. Jolly, *Zeitschrift f. rationelle Medicin*, Bd. xxxvi., 1869. (The same case is described by Nobiling, *Baier. Aerts. Intelligenzbl.*, 24, or *Jahresbericht für Medicin*, 1859, p. 153, quoted by Knox, *loc. cit.*)—Railway servant; died fifty-eight, of cancer of stomach. Mental power normal, brain of normal size, convolutions of both hemispheres well developed; corpus callosum length 2·8 cm. (about 1 inch); knee is 1·9 cm. thick; the body varied from 1·1 to 12 cm. thick; the posterior rudiment of the splenium 0·6 cm.; distance of knee from tip of frontal lobe 4·7 cm., of posterior margin from tip of occipital lobe is 8·5 cm. Psalterium of fornix absent, fornix present (rudimentary), ventricle dilated, ependyma thickened; anterior commissure apparently present, middle commissure absent, cornu ammonis normal. (It would have been interesting to know how far forwards it extended, and what was the condition of the fascia dentata and nerve of Lancisi.)

XXI. Chatto, *London Med. Gazette*, i., 1845.—Child, year old; epileptic (daily fits); in all its life manifested no sign whatever of recognising persons or objects. Corpus callosum represented by two thin strands, a few lines broad, uniting the anterior parts of the hemispheres; psalterium of fornix absent, septum lucidum also absent (fornix itself presumably present). No note of condition of other commissures. A small hyatid cyst, size of hazel-nut, lying anterior to corpora quadrigemina, with smaller ones adhering to it, containing gelatinous fluid; small quantity of fluid in ventricles; brain firm.

3. *Cases of Absence of Anterior Part of the Corpus Callosum.*

XXII.—Mitchell (Henry), *Med. Chir. Trans.*, xxxi. p. 239, fig. 14.—Boy, fifteen; civil and well conducted; slow in acquiring knowledge at school; could read and write, but in doing so had tendency to fall asleep; had difficulty in learning his trade, but was very shrewd in money matters; generally mentally sluggish. Injury to head from cricket ball three years before death (confined to hospital for twelve months). Brain and convolutions of normal size, skull and dura normal, anterior part of body of c.c. absent exposing ventricles, velum interpositum probably torn through, posterior $\frac{1}{2}$ of c.c. present, measuring $1\frac{1}{2}$ inch long, from anterior border to tip of frontal lobe = $3\frac{1}{4}$; posterior margin 2 inches from tip of posterior lobe; at side of cavity the corpus callosum persists as a thin rounded margin. The septa lucida, fifth ventricle, and most of the anterior pillars of the fornix were absent; anterior commissure and small part of the anterior pillars of the fornix, and most of the posterior part of the fornix were present. The radiating fibres from all parts of the corpus callosum seemed normal. Query? Was this not a case of dropsy of the fifth ventricle which had caused destruction of the anterior part of the c.c., the septa lucida, and the corresponding parts of the fornix? (fig. 14).

XXIII.—Langdon Down, *Med. Chi. Trans.*, xliv. p. 219.—Boy, aged nine; idiotic, could not stand, or feed himself, or speak; fond of music. Calvarium thick, somewhat unsymmetrical; brain weighed 2 lbs. 8 ozs. Membrane normal, velum interpositum present; posterior cornu of ventricles enlarged, positive absence of any septum lucidum; fornix present—its pillars widely separated; no commissure of body; anterior commissure present; two lines above it a transverse band (perhaps a rudiment of the corpus callosum) not more than $\frac{1}{16}$ in thickness; middle soft commissure absent.

XXIV. Langdon Down, *Med. Chir. Trans.*, vol. xlix., 1886, p. 195.—Male, forty. Could read easy words, learning to write a little, answer simple questions, fond of music, memory defective, fond of children, otherwise passionate. Died of pleuro-pneumonia. Calvarium unsymmetrical and dense, shelving anteriorly. On separating the two hemispheres the almost entire absence of the corpus callosum was apparent, and the velum interpositum exposed to view. A small cartilaginous-like band $\frac{7}{24}$ inch broad and $\frac{1}{24}$ inch thick, situated opposite the corpora striata, was the only representative of the great commissure. The fornix was represented by two thin posterior pillars; the body of the fornix and its anterior pillars absent. Right optic thalamus much larger than left; posterior cornu of lateral ventricles was distended with straw-coloured serum; pineal gland size of a wild-cherry; middle commissure absent.

4. *Cases where Absence of Corpus Callosum (or part of it) probably Secondary (to Hydrocephalus, Hydatids, or Tumours.*

XXV. Gausser, *Wiener Zeitschrift*, xi., 5th June, 1845.—Epileptic, twenty-six; central part of anterior half of the corpus callosum, also septum lucidum and anterior and middle parts of fornix, absent. Dropsy of fifth ventricle.

XXVI. Birch-Hirschfeld, *Arch. f. Heilkunde*, viii. p. 481.—Man, forty-one; of ordinary intelligence. Anterior half of corpus callosum absent; dropsy of third ventricle (and evidently the fifth) separating the two septa lucida; a cavity containing fluid in the left frontal lobe communicating with the third ventricle.

XXVII. Foerg, *loc. cit.*, pp. 17-25; see Sander, *loc. cit.*, p. 136.—Middle part of corpus callosum and body of fornix absent; otherwise everything normal.

XXVIII. Solly, specimen in St. Thomas's Hospital Museum.—Boy, sixteen; died seven days after fracture of skull. Mother says "he was never right from his birth" and supposed that his weakness of intellect was due to a difficult labour. He had always difficulty in controlling and regulating the action of his muscles so as to maintain the erect position, and was always stumbling and rolling about; he generally appeared drowsy; he was fond of reading (religious books being his favourites), but was unable to give a clear account of anything he had seen or read; childish in his amusements; he sometimes talked naturally, but was generally "boobyfied." Corpus callosum completely absent. A pale membranous bag protruded from left side, which on being cut into was found to be a cyst 2 inches in length and 1 in breadth, containing a serous fluid, and lined by a firm membrane. This formed roof of lateral ventricle on left side; the body and most of posterior pillar of fornix were absent; a portion of anterior column present. On velum interpositum was a small hydatid, and a considerable quantity of fluid in left and third ventricle. In the right ventricle everything was normal. Anterior commissure probably present; middle abnormally thick.

XXIX. Meierzejevski, *Revue d'Anthropologie*, 1876, No. 17; see Onufrowicz, *loc. cit.* 313.—Corpus callosum thin, anterior commissure absent.

XXX. Maclaren, *Ed. Med. Jour.*, 1879.—Female, aged thirty-two; imbecile, epileptic, deaf and dumb. Pia mater adherent along margins of longitudinal fissure; convolutions thin; white matter reduced; ventricles greatly dilated; septum lucidum absent; c.c. represented by two narrow belts—one at posterior, one at anterior extremity. Body of fornix absent; anterior and posterior pillars represented. Anterior, middle, and posterior commissures intact.

It is evident that the majority of the preceding cases are due to a primary arrest of growth and are only to be properly interpreted by the study of the development of the cerebrum. We learn from the work more especially of His and Mihalkovicz, that the anterior cerebral vesicle, which is primarily single, becomes at a very early period (about the eighteenth day) constricted in the middle line by the primitive falx cerebri, a process of vascular connective tissue. The two hemispheres thus formed grow up on either side of the falx, with their median walls at first plane and parallel to the latter; but during the second month there appear on them two curved fissures almost concentric with the free margin of the hemisphere (fig. 31, from Mihalkovicz). These fissures are termed respectively the fissura hippocampi (*f.h.*) (*ammons-furche*) and fissura choroidea (*adergeflechts-furche*). They begin anterior to the foramen of Monro, describe almost a semicircle over the corpora striata, and end near the tip of the temporo-sphenoidal lobe. The superior fissure forms a projection of the cerebral wall into the lateral ventricle, known as the pes hippocampi major, of which only the posterior part, that which projects into the posterior cornu, remains as a permanent structure. The inferior fissure, the fissura choroidea, is formed by the lateral outgrowth from the lower margins of the falx cerebri of the tela choroidea superior (velum interpositum) with its fringe of vessels, the choroid plexus. (See fig. 33, *falx*, *tel. chor.*, and *plex. chor.*) The cerebral wall covering this plexus becomes gradually reduced to the layer of epithelium, which forms its investment in the adult. The two fissures include between them a portion of the cortex (fig. 31, *r.b.* and *fasc. dent.*), which from its position and form is termed the convolution of the marginal arch (the randbogen of German authors). This convolution is continuous in front with that part of the cortex (*spt*) which forms the septum lucidum, and posteriorly it passes into the gyrus uncinatus. Along with the septum lucidum it becomes the seat of the following series of important changes:—

About the middle of the third month of intra-uterine life the triangular areas of the cortex which correspond to the two septa lucida (*spt*) become fused together and unite along their margins (thus including the cavity of the fifth ventricle between them).

In the beginning of the fourth month the lower borders of the fused septa lucida and of the as yet ununited marginal arches become differentiated into the anterior pillars, body, and fimbria (and commissure ?) of the fornix (fig. 31). About the same time (probably at a slightly later date) the anterior commissure appears in the lower angle of the septa lucida. Towards the end of the fourth month, along the anterior and upper periphery of the septa lucida, the rostrum and knee of the corpus callosum (fig. 32, *cal*) are developed. During this month also the two marginal arches become gradually united as far back as the posterior extremity of the optic thalamus.

During the fifth and sixth months the fused portion of the marginal arches becomes gradually differentiated from before backwards into the corpus collosum. With the exception of a small portion of grey matter (the induseum griseum), and the nervus Lancisii (*lnc.* fig. 32) above and of the fornix below the corpus callosum, the whole of this part of the marginal arch becomes modified into callosal fibres. In many mammalia the upper portion of the arch becomes collosal, while the underlying part becomes cornu ammonis, which thus extends much farther forwards than in man. The fusion of septa lucida and marginal arches necessarily causes the intercepted portion of the primitive falx to atrophy (fig. 33), so that the falx (*flx*) and tela choroidea superior (*tel. chor.*) become apparently two quite independent structures.

The portion of the marginal arches behind the point of fusion gives origin to the fornix (*fornix*), the fascia dentata (*fasc. dent*), and the nervus Lancisii (*lnc*). On its outer border is the fissura hippocampi (*f.h.*) proper; while the anterior part of this fissure now lying above the corpus callosum becomes the callosal sulcus (see Milhalkovicz, *Entwickelungsgeschichte des Gehirns*, pp. 120-130).

If we apply these facts to the study of the recorded cases of absence or partial defect of the corpus callosum, we find that the majority of these cases can be explained on the hypothesis of arrest of development, and that they may be classified according to the period at which this arrest takes place, the appearance of the brain varying accordingly.

1. *The Falx may constrict the Anterior Cerebral Vesicle, either not at all, or insufficiently.*—(Lesion occurs during first three weeks.) The cerebrum will consist of a single vesicle or of two imperfectly divided hemispheres, united by an unthinned septum (of grey matter). There will be one ventricular chamber, no tela choroidea superior, no convolution of the marginal arch, and therefore no fornix, no anterior commissure, and no corpus callosum. See cases recorded by Turner, *Journal of Anatomy and Physiology*, xii. p. 241 (fig. 29); Bianchi, *Storica del Monstri del Duo Corpi*, Torino, 1749, p. 100; Förster, *Missbildungen des Menschen*, 1861, p. 87, cases of Cyclopia; Hadlich, *Arch. f. Psychiatrie*, x. p. 99 (figs. 26, 26*a*, 27, 27*a*); and Wille, same volume, p. 597 (fig. 28).

2. *The two Hemispheres perfectly divided, but Septum Lucidum and Marginal Arch, if developed, fail to unite.*—There will be no anterior commissure, no corpus callosum, no psalterium of fornix. Tela choroidea superior continuous with falx cerebri. (Fornix present if marginal arch developed.) Development arrested before the fourth month. Cases II. (Ward), III. (?), IV. (?) (Foerg), XI., XV.

3. *Hemispheres formed, but Septa Lucida united only by Antero-Inferior Angle.*—Anterior commissure present. Other structures as in Class II. (Development arrested during fourth month.) Cases (several imperfectly recorded) I., V., VI., VII. (?), VIII., IX., X. (?), XII., XIII., and my case.

4. *Hemispheres formed; Fusion of Septa Lucida and Marginal Arches more extensive, but still incomplete.*—(*a*) Fusion limited to septa lucida. (Arrest of development at end of fourth month.) Anterior commissure and knee of corpus callosum present. Fornix present, but its psalterium absent (Case XXI.). (*b*) Union of septa lucida complete; but of marginal arches limited more or less to anterior part. Corpus callosum present anteriorly, but generally thin (as in lower mammalia). Splenium absent or thin. Psalterium of fornix present, if fusion has extended sufficiently far back. Cases XVI., XVII., XVIII., XIX., XX.

The destination of the septum lucidum and marginal arch in Series 3 (and in those cases of Series 2 in which they have been developed) remains to be examined. We have seen that these structures lie between the (embryonic) fissura hippocampi and the fissura choroidea, and that the fornix is developed along their inferior margin. If now we find a structure having the same relation or position to the fissura choroidea, the fornix and the fissura hippocampi, we may fairly conclude that it represents the septum lucidum and marginal arch. There seems little difficulty in identifying the area (*spt*) in my case (fig. 2), and in Onufrowicz (*spt*, fig. 16), Kaufmann (fig. 28), Anton (fig. 13), Eichler (fig. 11), and Knox (fig. 12), as the septum lucidum.

The marginal arch presents greater difficulty. Onufrowicz and Kaufmann consider that the fibres occupying its position belong to the system of fronto-occipital association fibres, and pass to the outer side of the posterior cornu of the lateral ventricle into the tapetum—a structure usually held to be composed of callosal fibres; that they are in fact the fibres of the cingulum of Burdach, no longer concealed by the fibres of the corpus callosum. This view I consider to be untenable, for the following reasons:—The cingulum lies in the substance of the gyrus fornicatus, separated by part of its grey matter from the corpus callosum (see Meynert, *Psychiatry*, p. 40, and fig. 18). The structure under consideration, however, is separated by a fissure from the gyrus fornicatus. In my case, its fibres certainly do not pass into the so-called tapetum, but seem rather to end in the investment of the cornu ammonis posteriorly (at least in their greatest part). And lastly, it does not become prominent in a brain in which the corpus callosum has atrophied (see fig 30, drawn from the brain sent me by Dr. Ruxton, pathologist of Wadsley Asylum, in which the anterior two-thirds of the corpus callosum had completely atrophied in consequence of a lesion affecting the centrum ovale of the frontal and part of the parietal lobes). Had this fronto-occipital association system been merely concealed by the corpus callosum, it should now be as prominent as in the cases of congenital callosal defect. It need not I think surprise us that this structure does not contain grey matter. We find what is undoubtedly septum lucidum contains only white longitudinal fibres, and in the fornix and nervus Lancisii we see the tendency to the formation of the marginal arch into longitudinal fibres. The causes of the arrested growth are very various, and must act at different stages of development. The principal factors concerned are the primitive falx and the septa lucida. Unfortunately, few of the records permit of our determining the cause in any given case, so that the hypotheses stated below are intended principally to aid future investigators. The causes may depend on—

1. The primitive falx cerebri—(*a*) its non-development during the first three weeks of life; (*b*) after its formation, its excessive resistance to atrophy, such as might result from intra-uterine leptomeningitis; (*c*) a permanently too deep position of the falx, such as might result from cranial deformity (Richter, *Virchow's Archiv*, 106). Richter considers that premature ossification of the basis cranii increases the angle between the two petrous temporal bones, and by thus stretching the tentorium cerebelli so depresses the free border of the falx that it divides the corpus callosum as it grows up against it.

2. Irregular distribution of the anterior cerebral arteries (Sander) passing between the septa lucida, and preventing their union.

3. Asymmetry of the hemispheres (resulting from asymmetry of cranium), so that the two septa lucida are not opposite each other.

4. Abnormal growths in the falx.

5. Nutritional disturbance in septa lucida, such as early hydrocephalus.

As causes of secondary defect are dropsy of the fifth ventricle (Mitchell Henry), hydatids, lesions in callosal arteries (Kaufmann and Eichler), in vessels of centrum ovale.

Several authors imagine that the area *rb* represents the stump of the corpus callosum, which has succeeded in growing so far toward the middle line. Von Gudden's law of the complete atrophy of a divided embryonic system seems to decide against this view.

The view of Professor Hamilton of Aberdeen with regard to the distribution of the callosal fibres, seems to be completely negatived by the appearance in my case and in those recorded by Onufrowicz and Kaufmann. It is obvious that if in the normal brain the corpus callosum is the main constituent of the internal capsule, the latter structure should almost disappear when the corpus callosum is absent. This however does not occur. In my case it was not possible to detect any abnormality in it; and Onufrowicz and Kaufmann make similar statements. Hamilton (*Proc. Roy. Soc.*, 1887), endeavours to explain this by the theory that the corpus callosum is present, but does not decussate—that it ascends to the cortex of the same hemisphere. Were that so the normal appearance of the tapetum should be present in the occipital lobe in my case. It is unquestionably absent. Further, in Buxton's case, fig. 30, where the anterior part of the corpus callosum is atrophied completely, sections taken at all levels show that the internal capsule is not in the least diminished. Buxton's case further serves to explain the apparent curving downwards of the corpus callosum into the internal capsule. The arched fibres remain though the corpus callosum is gone, but they are seen on naked eye and microscopic examination to come in very great measure from the gyrus fornicatus. It is no doubt the intermingling of the callosal and capsular systems that produces the appearance described by Hamilton. As further evidence of the separateness of those two systems may be mentioned the fact that in the mature human fœtus and infant up to three months, the callosal system is non-medullated; while in the mature fœtus the whole posterior limb, and in the three-months' child almost the whole of both limbs of the internal capsule are medullated. And further, in some of the lower mammalia the strand from the capsule to the gyrus fornicatus can be traced as quite distinct from the callosal system.

Lastly, the case is instructive with regard to the supposed functions of the corpus callosum. A great deal has been written as to its supposed function of co-ordinating the corresponding convolutions of the opposite hemispheres—a view which seems to date from Meynert's theory of its anatomical connections. It is right to state that Meynert's opinion is based on no proof whatever and the physiological view is equally speculative. It was supposed to account satisfactorily for the idiocy or imbecility of most of the cases. But examination of the literature shows that where there has been imbecility there has always been some other grave brain defect. On the other hand, the cases of Eichler, Paget, Malinverni, Jolly, and that recorded by me, and the second case of Kaufmann, and that of Erb (*Virch. Arch.*, 96), show that where the brain is otherwise well developed there may be "no disturbance of mobility, co-ordination, general or special sensibility, reflexes, speech, or intelligence, whether the defect of the corpus callosum be primary or secondary."

The radiated convolutionary arrangement is very difficult to explain. It may be due to the mechanical resistance offered by the ring-like marginal arch to the growth of the grey matter of the gyri. This will thus become furrowed much as a bag made of cloth when a string is tied tightly round its neck. In this case too, the furrows radiate outwards from the string. The abnormal mesial fissure of Rolando is not found in other cases. I am at a loss to account for it except on the view that the forward growth of the brain has surpassed that of the cranium, and that a duplicature of the inner surface was thus produced."

KATATONIA.

BY MM. T. SÉGLAS AND PH. CHASLIN.

Efforts are always being made to group together some of the numerous scattered facts existing in the wide field of insanity, in order to constitute distinct pathological forms. General paralysis remains till now the only undisputed one, although other attempts have been made, more or less justified. We intend to examine here one of those efforts, concerning which authors are not yet agreed, and which has been described in Germany as katatonia or *Spannungs Irresein.*

I.

The first and principal work on katatonia dates from 1874 and is due to Kahlbaum[1] who tries in an important monograph to define a form of disease in which certain physical, and more particularly muscular symptoms accompany (as in general paralysis, and as frequently) certain psychical phenomena, and play a leading part in the whole morbid process.

This new form of mental derangement may be closely allied to melancholia attonita, which is ordinarily considered to be a distinct disease. On careful examination of cases of the latter disease we can very often according to Kahlbaum discover at the onset epileptiform seizures or other manifestations of spasmodic attacks. These conditions become permanent, attain their greatest development in the *flexibilitas cirea* stage of the mental condition and merge into the final stage of dementia. These symptoms are by their importance placed on a line with the paralytic phenomena of general paralysis. By their side, and in addition to the usual symptoms of melancholia attonita, we find other physical, and more especially psychical, phenomena, notobly a particular form of exaltation, which may be termed "pathetic ecstasy," as well as a tendency to speak as if discoursing or to recite, which gives a characteristic physiognomy to the disease. All these symptoms constitute what is called Katatonia, and up to a certain point this form of disease should be considered as a counterpart to certain forms of general paralysis with or without grandiose delusions. Analogous to general paralysis as regards the succession of the different psychical phenomena in connection with the muscular symptoms, they seem to differ from it, on the contrary, by the quality of the muscular and psychical manifestations, and consequently a marked difference is to be found in the prognosis.

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[1] Kahlbaum, *Die Katatonie*, Berlin, 1874.

If we study all the psychical phenomena of katatonia, it will be found that this disease exhibits in succession the chief forms assumed by diseases of the mind, such as melancholia, mania, stupor (*attonität*), intellectual enfeeblement, and finally dementia. The intellectual enfeeblement is generally accompanied by delusional conceptions, which are active but badly arranged, and often even incoherent (*Verwirrtheit*). The duration of each state is very variable, and frequently we find alternations of depression and excitement, but on the whole, the melancholic stage is of longest duration. Melancholia with stupor either follows immediately upon the primary melancholia, or is more frequently separated from it by an attack of mania. It should be remarked that in the cases where this attack of mania is absent there has often been some such outburst of excitement at a previous period of the patient's history. In very rare cases the disease commences with a condition of stupor, and this happens chiefly where there have been violent physical or moral shocks, such as an intense fright, injuries, hanging, &c. At other times excitement succeeds on an attack of stupor of short duration; or it may be a condition of intense melancholia followed by a state of stupor with or without a period of maniacal excitement, which latter may be considered as a further development of the morbid process.

Sometimes in the midst of a long period (weeks or months) of mania there appear only a few days of stupor. In other and rarer cases the stupor alternates with a condition of speech-incoherence. The terminations may be dementia, recovery or death. There are also cases where the katatonia develops itself in the midst of a condition of nervous excitability or of general physical depression, but then the katatonia does not commence with a period of melancholia (unless a mild attack of hypochondria be so termed), but with a period of mania.

In the majority of cases the stupor lasts longer than any of the other periods, but it must be noted that the transition to dementia takes place in an imperceptible manner.

However it may be, the condition always presents a marked cyclic character. Usually it commences slowly and progressively, later on the katatonia attains to a condition of crisis, and lastly it abates, passing into a condition of dementia. In many cases a state of general verbal confusion develops itself after the stage of stupor and before the complete dementia, and this stupor is preceded by a maniacal period separating it from the initial melancholia. The stupor may therefore be considered to indicate the onset of the period of decrease.

As regards the particular symptoms, they present nothing very special, except those of the stage of exaltation. As a whole there is either agitated melancholia, or the most violent excitement, or else a more regular and systematised delirium (*Wahnsinn*). We then meet certain symptoms peculiar to katatonia which enable us to make the diagnosis even antecedent to the period of stupor. First there are the *pathetic characteristics*, in the form of theatrical exaltation, and of tragico-religious ecstasy, in which the patients both speak and act. They recite and make speeches perpetually, gesticulating all the time, arriving often even to the idea that they wish to become actors, or even that they have already attained to their ambition. They utter the most commonplace remarks as if they were convinced that those expressions were of supreme interest to mankind, or they speak of most serious subjects much above their knowledge, and without expressing well-defined grandiose delusions, they believe at last that the world is particularly interested in the trifling events of their existence. The mania of reading, speech-making, loudly reciting (*Redesucht*), which one meets during the stage of exaltation is very different to the senseless words and cries of the ordinary maniac. Among the other characteristics peculiar to katatonia we notice a tendency to the repetition of words and phrases without any meaning, and without following each other in proper sequence, but pronounced as if the patient were holding a discourse. This "verbigeration" is a co-ordinated spasm of the muscles of speech originating in the cerebral speech centres, and is absolutely special to katatonia. It must not be confounded with the idle, senseless talk of the ordinary loquacious dements (*Verwirrten*), and mentally enfeebled, with the ordinary reciter, with the person of flighty ideas (*Ideenflucht*), or with a condition of *confabulation*.[1] In the course of the disease however the verbigeration can be transformed into any of these other forms; moreover, along with the verbigeration we must note the remarkable habit of frequently using diminutives. With regard to the dumbness of the stage of stupor, it may be absolute or relative, partial or intermittent. Sometimes it is voluntary (owing to the fear produced by a delusional idea, or a hallucination), at other times it is involuntary (from an absence of ideas and incapacity of attention), and in other cases the condition remains inexplicable. Whilst the speech-making loquacity (*Redesucht*) is attributable to a clonic convulsion, the dumbness seems on the contrary to be due to a tonic convulsion.

Frequently during the period of stupor there seems to be an entire absence of the formation of ideas, a cessation of the thinking processes; at other times one may discover, on the one hand, delusional ideas with hallucinations, such as are found in melancholia, and on the other hand ideas of grandeur, such as of illustrious birth. Again, some patients are not a prey to depressing thoughts during the stage of stupor, but on the contrary, they seem to have from time to time amusing impressions, as is shown by their occasionally laughing. Ideas of a religious or erotic character are said to be very frequent.

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[1] "Confabulation" is distinguished from "verbigeration," by its character of creative and phantastic imagination (*phantastisch-productiver Inhalt*). Kahlbaum; *loc. cit.*, p. 39.

It is likewise to be noted that in katatonic insanity there is a condition of monotonous movement and a resistance offered to any interference, especially when one attempts to give another direction to these movements. The katatonic patient likes to remain in bed and even refuses food during the stage of excitement—sometimes from a fear of any change in position or movement, but generally without reason, whether delusional or not. Further, there is a certain tendency to make stereotyped gestures, or to assume ridiculous attitudes, to make peculiar grimaces, more particularly puckering the lips (*schnauzkrampf*), even from the beginning of the disease, or during the remissions when delusions are no more apparent.

As regards the physical symptoms, they are all based upon a disturbance of the motor nerves. Very frequently, and especially during the stage of stupor, we may observe a relaxation of the limbs; we may also find, even from the beginning of the disease, choreiform convulsions, epileptiform, or tetanic spasms. These convulsions may be general or partial in their distribution. The convulsions whether clonic or tonic must be ascribed partly to the psychical state, and partly to the physical condition. We never find real motor paralysis: we may find anæsthesia, more or less complete, apparent or real; hyperæsthesia is frequent, and localised occipital cephalagia is pretty characteristic of katatonia.

Amongst the other physical phenomena let us notice the frequency with which the legs are swollen; sometimes there is œdema of the eyelids; there is abundant cutaneous desquamation, marked anorexia, the breath is foul; there is disturbance of the gastro-intestinal functions, and, finally, chlorosis is not uncommon. We must also note the importance and excessive frequency of phthisis in this disease—rare according to Kahlbaum in other mental conditions. This last fact will have to be opposed to the predominance of pneumonia in general paralysis.

According to Kahlbaum the ætiology of katatonia offers nothing very particular; heredity seems to have very little influence in its production, and sex does not influence its frequency, but the disease is most prevalent in youth and adult age. Sexual excesses and onanism in youth seem to create a serious predisposition to it. With women we might attribute the cause either to pregnancy or to the puerperal state. Overwork and excessive religiousness seem to play an important part in the causation, and we count among those subject to katatonia a large number of schoolmasters, the sons of schoolmasters and theologians. Further, anæmia and a general condition of nervousness seem to occupy the first position among the predisposing causes. The occasional causes are those of the other forms of mental disease; we must not omit however to refer to certain forms of traumatism, more especially hanging, as occupying an important ætiological position. It would also seem to us that imitation plays a part in the ætiology, from Kahlbaum's point of view, for he classifies under katatonia the epidemics of the convulsionists and preachers (Suède).

The prognosis of this affection is upon the whole favourable, and in this again it differs from general paralysis: habits of antecedent self-abuse seem to aggravate the disease. Apart from tubercular complications, katatonia may cause death; if cured, a relapse never occurs, and it does not exercise any hereditary tendency in the children of persons who have suffered from it. However, let us remark in passing, the author seems to us to have little faith in degeneration in general.

With regard to the pathological anatomy, Kahlbaum reports very extensively the results of seven autopsies of persons subject to katatonia, and he institutes a comparison between the pathological conditions found in his cases and those found in general paralysis. There seems to be in the early stages of the disease a condition of general stasis in all the cerebral vessels, along with serous effusion, which produces softening of the cerebral tissue without retraction or shrinking, but with the formation of exudation both on the coverings and ventricles. This exudation affects chiefly the arachnoid membrane, and is especially to be found at the base of the brain. In old cases one finds some shrinking of the cerebral tissue, and the exudation partially organised. Contrary to what happens in general paralysis there are ordinarily no meningitic hæmorrhages, but the arachnoid is regularly the seat of pathological changes. In the cases where death has occurred at an early stage, the arachnoid was opaque over the pons, and the opacity extended over the cerebellum to the medulla oblongata immediately behind the fourth ventricle. In the other cases the arachnoid was found to be thickened in the same regions; further, there was a remarkable tendency for serum to exudate in the neighbourhood of the base of the brain, which accounts for the marked diminution in the number of the Pæchionian granulations.

According to Kahlbaum mental diseases in general are caused by disturbances in the nourishing processes which, commencing with hyperæmia and exudation, terminate in atrophy, dropsy, and finally the formation of new tissues. Katatonia acts in the same manner but with this difference, that the stasis is essentially transitory and weak in the early stage; the shrinkage and atrophy set in much later, thus preventing due dilatation of the cerebral cavities, contrary to what takes place in general paralysis. Moreover, katatonia has a predilection for the arachnoid and for the base of the brain, the exudation extending itself to the Sylvian fissure and towards the second and third frontal convolutions. This latter fact is of great importance in explaining the dumbness as well as the verbigeration. It must be added however that the arachnoid was not in every case altered in these special regions, and besides, the pia mater was not adherent to the subjacent convolutions in the majority of the cases.

An examination by the microscope has produced no results. Kahlbaum admits himself that these data are a little insufficient, but that they form a point of departure for future researches.

Pulmonary and intestinal tuberculosis are found very frequently. These may be complications, due secondarily to the katatonia itself, the muscular rigidity producing imperfect respiratory action which might permit tuberculosis to develop itself in lungs insufficiently distended with air.

To sum up, katatonia is a cerebral disease, the characters of which change in a cyclic manner in such a way that the psychical symptoms present successively the forms of melancholia, mania, stupor, loquacious dementia (*Verwirrtheit*), and ending finally in complete dementia. To this picture of the whole disease there may be wanting one or more symptoms, but on the other hand there may appear, as the chief phenomena, affections of the neuro-motor system presenting the general character of muscular contractions. This form of disease thus distinguished approaches general paralysis with or without grandiose delusions, especially when considered from its clinical aspects. In general paralysis there are also symptomatic indications which change in a cyclic manner and are accompanied by disturbances of the motor-nervous system; here however having the characters of paralysis.

Closely allied to these two forms of such well-marked diseases we have to arrange a third form. In this latter the symptomatic evolution is equally typical; but when we consider the neuro-motor system we find an entire absence of symptoms. This last form which is frequently met with in asylums, exists as mania and it often terminates in recovery, and when it is contrasted with some complicated maniacal forms it may be termed simple or veritable mania.

To these forms pursuing a cyclic course we must oppose all the cases in which the symptomatic whole remains unvaried (partial insanities), and those in which the symptoms are changing and the course not cyclic (ordinary sympathetic, febrile and traumatic insanities).

Katatonia is not a partial alienation, but it includes among its symptoms, more or less, all psychical manifestations. It does not develop itself after physical diseases, but rather on a predisposed ground (anæmia), and, by its cyclic and typical course it differs from idiopathic and sympathetic forms of mental alienation.

One may distinguish epileptic, tetanic, choreic, cataleptic and indifferent forms of katatonia; but it is better to classify them as weak or simple cases, grave or complicated. Among the first class we may place melancholia attonita, which is already isolated as a separate affection in ordinary forms of classifications of mental disease, but which according to Kahlbaum may be termed a form of mild katatonia. For, during the condition of stupor one may always recognise some neuro-motor symptoms as well as convulsive attacks previous to the admission of the patient to an asylum, and which the doctor fails to notice; and similarly there are frequently found short periods of excitement—a sort of passing mental exaltation—interrupting the ordinary course of the melancholic condition; but with such rapidity and in such a transitory manner, that the aspect of the melancholia does not seem to be altered by it.

A second group may comprise cases in which after a melancholic beginning, mania sets in more or less pronounced, more or less durable, and which, disappearing before the stage of stupor appears, have been mostly described as cases of simple mania. Next come the cases where one notes neuro-motor symptoms of long duration, and so well marked that the medical attendant regards them as curiosities and complications without regular character (K. gravis). Finally, in the last form (protracted katatonia) the symptoms of excitement do not appear in the first half of the disease, but in the later stage, and mostly in the form of remissions and intermissions.

The diagnosis of this disease would be after all easy, and this is what Kahlbaum says of it: An isolated case of convulsions, considered as epilepsy, eclampsia, apoplexy, meningitis, or encephalitis, which appears in a condition of complete health or of mental trouble of a certain duration, and which (without determining paralytic phenomena) is complicated by excitement or intense emotional depression, leads invariably to a condition of dumbness without motive or at the very least to a cataleptic state. Lastly, it may be complicated by symptoms of resistance. If no recovery takes place a state of stupor will supervene. Or, perhaps if we find a marked pathetic expression and an attitude of peculiar stiffness in a patient who is acutely melancholic, we may be able to predict almost certainly the commencement of stupor (attonity). Again, if a patient who used to speak leaves off doing so and that permanently, the head and limbs being in a rigid condition, this disease is certainly katatonia. In the absence of further data one might confuse the apathy with the stiff and rigid habits of infantile dementia, or the evanescent mental outbreaks following physical diseases.

Only in two cases is the diagnosis really difficult. The first case is when in the commencing half of the disease the dumbness is not continual, and the neuro-motor symptoms have not yet appeared. The pathetic attitude, and the obstinate repetition of a word are then characteristic. The second difficult example will be in a case where melancholic symptoms without neuro-motor manifestations have existed for a long time, and where dumbness develops itself without muscular stiffness at all, and without alteration in the ordinary melancholic symptoms. The important symptoms for purposes of diagnosis will then be found in the repetition of a word or discourse, gesticulations and stereotyped attitudes, obstinacy and resistance.

Since the publication of the work of Kahlbaum we find a certain number of other authors who have written on the subject, some admitting, at least in the main, the descriptions and conclusions of Kahlbaum, and others criticising them. It is with the first class of authors that we will now occupy ourselves.

Hecker[1] reports in support of the opinions of Kahlbaum two cases of katatonia, to which however serious objections might be offered, and accordingly we will have to refer to them again later on. Brosius,[2] with regard to katatonia, insists upon the importance of the verbigeration, the absence of sudden changes in the emotional state, and the amnesia which follows the agitated periods. There would be no real mania in katatonia during the period of stupor; there is rarely a condition of emotional depression, but frequently a kind of ecstasy or general indifference. He believes that we can already distinguish three forms of katatonia :—

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[1] Hecker, *Allg. Zeitsch. f. Psych.*, 1877, Bd. xxxiii., p. 602.

[2] Brosius, *Die Katatonie* (*Allg. Zeitschr. f. Psych.*, 1887, Bd. xxxiii. *S.* 770.)

(1) *A meningitic form*, the prolonged course of which is connected with the residue of meningitis.

(2) Another form connected with *cerebral anæmia*, such as one sees frequently in the clinique, when the amelioration in the symptoms of katatonia runs parallel with that of the general health. Lastly there is

(3) A third form, namely, cerebral *œdema*, which has been described by Etoc-Demazy.[1]

Kiernan[2] in two consecutive memoirs on this subject reproduces in substance the ideas of Kahlbaum. He refers particularly to the heredity of the strumous diathesis, to the facility of simulation on account of the regularity of the symptoms of the disease. He writes much about the pathological anatomy, and confirms by his own autopsies those of Kahlbaum, which show old but cured hydropsy and some basilar meningitis. The deductions of Meynert in Kahlbaum's cases lead him to think that the disease has been preceded by a meningitic process, localised to the base of the brain or over the fissure of Sylvius. According to Kiernan, katatonia is frequently preceded in infancy by basal meningitis of a tubercular character, extending itself likewise to the Sylvian fissure and fourth ventricle. With regard to this meningitis, he refers to the opinions of some authors as to the origin of the motor symptoms, convulsive or otherwise, accompanying the basal meningitis. He notes besides in passing that Meynert described katatonia as a particular form of melancholia attonita two years before Kahlbaum, and he cites the opinions of Meynert regarding the pathology and physiology of this affection—certainly very ingenious ideas, but they are perhaps a little hypothetical. In addition, he gives details of an autopsy, followed by microscopic examination, in which he again finds the remains of the tubercular meningitis, and a condition of venous statis of vaso-motor origin; no other alteration was found except slight sclerosis of the white matter of the spinal cord. He finds a great analogy between the state of the brain one observes in this condition and that found in cases of typhoid fever, but the essential and characteristic pathology of the disease is, according to Kiernan, a primary disturbance in the vaso-motor centres, producing sanguineous stasis, and this he maintains is the point of departure of the whole morbid process.

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[1] Etoc-Demazy, *Th. de Paris*, 1838.

[2] Kiernan, *Alienist and Neurologist*, 1882; *Detroit Lancet*, 1884.

Hammond[1] describes katatonia as a special form of mental disease distinguished by alternating periods presenting themselves with more or less regularity of mania, melancholia and epileptiform and cataleptiform states, with primitive delusions of an exalted character and with a dramatic tendency. But after all this author adds nothing to the description of Kahlbaum, whose ideas he accepts.

Spitzka[2] classifies katatonia among the group of genuine mental diseases not being the essential manifestation of a neuropathic constitution, nor having any relation to the biological epochs. It figures in the class of diseases without demonstrable lesions of the brain, amongst those which are *primary insanities*, and characterised by expansive, fundamental or emotional symptoms (mania), depressive (melancholia) or pathetic (katatonia).

Neuendorff[3] reports two observations which have been communicated to him, but presenting certain deficiencies, and which he compares to the katatonia of Kahlbaum, after a rather confused discussion, and without arriving at a very precise conclusion.

Schüle[4] devotes a whole chapter of his book to katatonia and seems to differ in his opinions regarding it from those authors we have already referred to. According to him katatonia is a special form of systematised acute hallucinatory insanity[5] (Wahnsinn), characterised by a nervousness of the muscles which are tense; this is sometimes permanent, at others intermittent or irregular, whilst at the same time the conscience of the individual—a victim to hallucinations and illusions—closes itself more or less completely to the influence of external perceptions. The motor rigidity may adopt a physiognomonic character, and as such express in a realistic manner an insane idea (such as the attitude assumed by a pugilist, a preacher, or one undergoing crucifixion) or something which may be purely physical (cataleptic or tetanic). The psychical stage may limit itself to the condition of acute insanity, or descend to the condition of real temporary dementia (stupor). Recovery is possible in both cases, but in the last it takes place after a period of marked intellectual weakening, with katatonic reminiscences at intervals. The course of the disease is cyclic and accompanied by a significant implication of the vaso-motor system which converts it into a true psycho-neurosis. The varying states of excitement depression and rigidity which one meets, seem (when their relations and order of succession are considered) to be connected with the course of the vaso-motor nerves. From its clinical aspect this form of insanity may be divided into expansive or depressive katatonia (with religious or demoniacal delusions), and further, into katatonia resting upon a basis of hysteria. While treating of hysteria, Schule reverts to this subject and amongst the forms assumed by hysterical insanity, he describes katatonic wahnsinn. This very frequent form of hysterical wahnsinn generally commences in maniacal excitement; then there always arises the question of an "invalid" constitution (by birth or acquired) which is mostly associated with anæmia. The importance of puberty, self-abuse, &c., must also be noted.

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[1] Hammond, "Remarks on Cases of Katatonia" (*Amer. Journ. of Neurol. and Psych.*, 1883, p. 302).

[2] Spitzka, *Amer. Journ. of Neur. and Psych.*, 1883, p. 313.

[3] Neuendorff, *Centralblatt f. Nervenheilk.*, 1883, No. 23, p. 529.

[4] Schüle, *Klinische Psychiatrie. Specielle Pathologie und Therapie der Geisteskrankheiten* (Leipsig, 1886).

[5] A subject written upon by M. J. Séglas (*La Paranoia. Arch. de. Neur.* 1887).

A quite recent work by Clemens Neisser[1] also appears to support the existence of the disease which is now under our consideration. Neisser only admits the katatonia of Kahlbaum and rejects the description of Schule, who refers to it as a form of systematised mania (*Wahnsinn*). For according to him the motor difficulties are primary and fundamental; the physical modifications being secondary and subordinate, and otherwise of little importance, as is indicated by Roller.[1] He admits also that the stupor is not necessarily a symptom of melancholic depression, but an essential part of the motor phenomena of katatonia and of that alone. While desiring to remain exclusively on the clinical and even "empirical" domain of description, he ventures to give physiological explanations as well, and he attempts—in following the ideas of Rieger[2]—to refer the cataleptic and other phenomena, the indications of opposition, &c., to the same cause, namely, the pathological innervation of the muscles antagonistic to those which must accomplish a given act. He reports besides a certain number of interesting observations which lead him to conclude that it is not merely some certain or special symptoms but the whole disease which constitutes a striking spectacle "to the eye and tact" of the clinical student, and this whole is katatonia. Neisser neither refers to the differential diagnosis, the etiology, or the pathological anatomy of the disease, and he does not occupy himself at all with the foundation on which it can develop itself.

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[1] Clemens Neisser, *Ueber die katatonie*, Stuttgart, 1887.

II.

On the other hand, by the side of these authors who admit the existence of katatonia,[3] there are others who express entirely opposite opinions.

Arndt[4] rejects the existence of this disease as an essential form of *Spannungs-Irresein*.

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[1] Roller, *Ueber motorische Störungen beim einfachen Irresein* (*Allg. Z. f. Psych.*, Bd. xlii., *H.* 1, 1885).

[2] Rieger, *Ueber Normale und Kataleptische Bewegungen* (*Arch. f. Psych. und Nerv.* Bd. xiii., 2, 1882).

[3] We may be permitted to refer in this place to several monographs on katatonia, which we have been unable to procure: Rush, *Diss. inaug.* 1879; Rebs, *Ein Fall von katatonie*, Diss. Erlangen, 1877; Jensen, *Allg. Encycl.* Bd. xxiv. At one of the last meetings (Nov. 2nd, 1887) of the Medical Society of Berlin, referring to a communication by M. Moll, on "Hypnotism," M. Jensen described the katatonia of Kahlbaum, which he classed after the epileptic or unconscious states, and which, he said, closely resembled hypnotism (*Deutsche mediz. Zeit.*, Nov. 10th, 1887, p. 1026).

[4] Arndt, *Ueber Tetanie und Psychose* (*Allg. Z. f. Psych.*, 1874, Bd. xxx., S. 28) and *Ueber Katalepsie und Psychose* (*Ibid S.* 58).

Westphal[1] admits, as does the original describer of katatonia, that the stupor is not necessarily accompanied by melancholia, and that it is sometimes found in delusional insanity (*Verrücktheit*) with most marked delusions of grandeur, but that katatonia is not a special form of insanity; it is only an acute and somewhat peculiar form of *Verrücktheit*, in which the motor symptoms do not possess the character of spasms, neither have they the importance which Kahlbaum wishes to ascribe to them.

Tigges[2] gives to the assembly of German alienists at Nuremberg in 1877 statistics of various cases of mental alienation, in which he finds symptoms which one may attribute to katatonia. He does not at all admit such a distinct form, and to him the stupor and other special katatonic phenomena are only symptoms.

Von Rinecker[3] read to the assembly of alienists of 1880, at Eisenach, a work by Fink on hebephrenia. In this memoir Fink describes three cases which according to him resemble very closely katatonia, and exhibit nearly all its symptoms. He cites Hecker (*Allg. Z. f. Psych.* Bd. xxxiii., S. 612), who describes a case of katatonia followed by hebephrenia. But according to Fink although katatonia is of a favourable prognosis, the insanity of puberty is very serious. A discussion took place on this subject. Sander did not admit the existence of hebephrenia and opposed Hecker's interpretation of it. Mendel rejected at the same time the insanity of katatonia and that of puberty. Sander replied that these attempts were detrimental to a satisfactory classification, and finally Rinecker declared that while he admitted the existence of hebephrenia, he refused to accept the existence of katatonia.

Krafft Ebing[4] makes a variety of "folie circulaire" of

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[1] Westphal, *Ueber die Verrücktheit*, (*Allg. Z. f. Psych.*, Bd. xxxiv., 1878, *S.* 252.

[2] Tigges, *Kahlbaum's Katatonie* (*Allg. Z. f. Pysch.*, Bd, xxxiv., 1878, *S.* 731.

[3] Rinecker, *Ueber die Bedeutung der Hebephrenie, &c.* (*Allg. Z. f. Pyschiatrie*, Bd. xxxvii., *S.* 570). Fink, *Beitrag zur Kenntniss, &c.*, *id. S.* 490.

[4] Krafft Ebing, *Lehrb.*, 2nd ed., vol. ii.

Kahlbaum's katatonia. Tamburini,[1] at the fifth congress of the Psychological Society of Sienne, in September, 1886, described some observations of katatonia and melancholia attonita with cataleptic phenomena. He asks himself whether the cases reported as typical, really ought to constitute a special morbid condition, because katatonic phenomena are to be found in other diseases, and because its course is that of a typical example of delusional insanity; thus bringing it under the forms accepted in our existing classifications, and he is inclined to consider it as a form of "folie circulaire" with katatonic phenomena.

These are all the principal attempts which have been made to isolate katatonia from closely allied forms of insanity. We have been much struck by the differences which exist between authors, not only in point of detail, but even in the manner in which the disease is considered, when taken in its entirety; the opinions of Schüle more particularly, varying considerably from those referred to in the memoirs we have just analysed. We have also seen that many authors totally rejected the conception of katatonia, and we will cite still others who without offering any opinion whatever on katatonia nevertheless report analogous cases, which they describe under different names. Moreover, even antecedent to Kahlbaum's memoir,[2] numerous cases of katatonia, although not specified, were to be found in special books; these cases are classified under mania, melancholia or stupor, as the special katatonia phenomena impressed the observers as merely complications of the disease they were describing (Hardy,[3] Clevenger,[4] Burrow,[5] Kelp,[6] Guislain,[7] Griesinger,[8] Morel[9]).

But even according to the opinion of the advocates of katatonia, it was under the name of stupor that the affections had for a long time been diagnosed and described, especially in France (Baillarger[1]). At the present time also, even after the work of Kahlbaum, many authors continue as in the past to report these symptoms as merely varieties of other diseases.

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[1] Tamburini, *Sulla Catatonia* (*Riv. sp. di fren*, 1886).

[2] According to Hammond (*loc. cit.*) one of the first cases of katatonia is to be found in the reports of Bethlem Hospital.

[3] Hardy, *Am. Jour. of Neur. and Psych.*, vol. iii.

[4] Clevenger, *Ibid.*

[5] Burrow, 'Commentaries,' 1828.

[6] Kelp, *Corresp. blatt. f. Psych.*, 1863, p. 357, and 1864, p. 322.

[7] Guislain, *Leçons orales sur les Phrénopathies*, 1852.

[8] Griesinger, *Traité des malad. ment.* (*trad. franç. de Doumic*, 1865).

[9] Morel, *Études cliniques*, vol. ii., p. 275 and following, 292, 293.

M. Cullerre[2] has published a description of catalepsy in a case of hypochondria with delusions of persecution, which we find mentioned by German authors. Further we have the description by Lagardelle[3] of a case of catalepsy following an attack of acute mania. These observations in our opinion remind us slightly of the katatonia of Kahlbaum.

M. Dagonet[4] seems to connect these facts with stupidity. The same ideas are found expressed in the recent work of Krœpelin.[5] Amongst the observations of melancholia with stupor, and cataleptic phenomena, published under this title since the memoir of Kahlbaum, and which we have been able to procure, we may mention those of Angelucci,[6] Wiglesworth,[7] J. Voisin,[8] and Wagner.[9] In other analogous cases (J. Adam[10] and Sankey[11]) hysteria seems evident but has not been pointed out specially. In another similar case Fritsch[12] insists upon the importance of hysteria and degeneration.

The influence of degeneration is also admitted by Maudsley[13] who while speaking of hebephrenia gives a description comparable to that of katatonia, which has been quoted, moreover, even by the advocates of the latter.

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[1] Baillarger, *Ann. med. Psych.*, 1843 and 1853.

[2] Cullerre, *Ann. med. Psych.*, 1877, p. 177.

[3] Lagardelle, *Ann. med. Pysch.*, 1871, p. 38.

[4] Dagonet, *Traité des Malad. ment.*, 1876.

[5] Krœpelin, *Comp. der Pysch.*, Leipsic, 1888.

[6] Angelucci, *Lo sperimentale*, May, 1880.

[7] J. Wiglesworth, "On the Pathology of Certain Cases of Melancholia, Attonita or Acute Dementia" (*Jour. Ment., Sc.*, 1883, p. 355).

[8] J. Voisin, "*Notes sur un cas de mélancolie avec stupeur à forme cataleptique, &c.*" (*Arch. de Neur.*, 1877, vol. xiii., p. 354).

[9] Wagner, *Anal. in Semaine medicale*, 6th July, 1887, p. 280.

[10] J. Adam, "A case of melancholia with stupor and catalepsy" (*Journ. of Ment. Sc.*, 1884, p. 508).

[11] Sankey, 'Lectures on mental diseases,' 2nd ed., 1884, p. 208, case 13.

[12] Fritsch, "*Zur kenntniss der melancholia attonita*" (*Wiener med. Presse* 1878, p. 1477, 1512, 1574).

[13] Maudsley, *Pathologie de l'esprit* (*trad. franç. de Germont*, 1888, p. 478).

Lastly, in his book on 'Folie à Double forme,' M. Ritti[1] refers to the presence of cataleptic symptoms during the melancholic stage and reports some observations on it. Several are taken from the writings of Kraft-Ebing whose views the author seems therefore to adopt.

III.

To sum up, we find ourselves before two opinions: (1) either katatonia is an essential morbid form, or (2) the cases classed under that name are only variations of types already known and described. Let us in the first place examine opinion number one, namely, that katatonia is a special form of disease.

Amongst the characters given as pathognomonic, we observe figuring in the front rank katatonic phenomena of the most varied nature: the pathetic attitude, stereotyped gestures, verbigeration, marked obstinacy (often systematic) and finally the cyclic course of the disease, on which however some authors insist less than others (Neisser).

Here a question arises. Are these katatonic phenomena as well as others mentioned before, really characteristic of a special form of mental disease? Let us consider them first by themselves and individually, beginning with the most important ones—the katatonic symptoms properly speaking.

A.—As one can see from the description of Kahlbaum, these symptoms may be most numerous, and of the most variable character—spasms, general or partial, epileptiform, hysteriform, choreiform, or tetaniform convulsions, cataleptic conditions, or even a condition of simple muscular stiffness. Together we here meet, isolated or even united in the same individual, almost all the known possible disturbances in the domain of the neuro-motor as well as the muscular systems (with the exception, however, of paralyses). Without considering the cases where these symptoms can show themselves in connection with different forms of disease (*e.g.*, rheumatism, typhoid fever), we can see that each of them may be met with in the most varied psychopathic conditions (Arndt, Krafft Ebing,[1] Freusberg,[2] Edel[3]).

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[1] Ritti, '*Traité clinique de la folie à double forme*,' 1883, Obs. 11, 12, 13.

In the first place we can classify the hysterical psychoses. The insane hysterical patient remains none the less hysterical; the insanity does not suspend itself, nor is it replaced by a fatal termination, such as may be produced by physical manifestations of nervous lesions, as convulsive, choreiform or tetanic seizures. So also with convulsions or contractions, cataleptic conditions are very frequently associated with hysteria apart from the attacks of catalepsy and hypnotism. Indeed, if in the state of watching the hysterical patient can exhibit the neuro-muscular hyper-excitability of lethargy or the cutaneo-muscular hyper-excitability of somnambulism, then we may also find the muscular flaccidity of catalepsy. Lasègue[4] had already noticed the fact of catalepsy existing during the state of being awake, in the case of hysterical patients. M. Charcot[5] in his lessons on hystero-traumatic paralyses speaks of a patient subject to hysteria, who presented while awake cataleptic immobility of his limbs, even when placed in the most varied positions. MM. Binet and Féré[6] have recently taken up these studies in an interesting memoir, and have reported new instances as examples of this condition of muscular plasticity while awake.

These phenomena may also exist in degenerative conditions with mania, especially in the mystic (Morel[7]) or erotic types, and even apart from all maniacal symptoms; for example, we find it with imbeciles and epileptics. We shall here report two examples which we have been enabled to study while under the care of our excellent colleague, M. Ch. Féré, of Bicêtre.

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[1] Krafft Ebing, *Lehrbuch*, B. 1.

[2] Freusberg, *Ueber motorische symptome bei einfachen Psychosen* (*Arch. f. Psych.* Bd. xvii., 1886, *S.* 757).

[3] Edel, *Allg. Z. f. Psych.*, Bd. xliii., 1886.

[4] Lasègue, *Catalepsies partielles et passagères, in Etudes médicales*, vol. i., p. 899. *Anesthésie et ataxie hystériques, Ibid.*, vol. ii., p. 85, and following.

[5] Charcot, *Leçons sur les maladies du système nerveux*, vol. iii., p. 357.

[6] A. Binet and Ch. Féré, *Recherches expérimentales sur la physiologie des mouvements chez les hystériques*, in *Arch. de physiol.*, Oct. 1, 1887, p. 323.

[7] Morel, *Traité des mal. ment.*, 1860, Note of page 491, and *Etudes cliniques*, vol. ii., p. 178.

CASE I.—L., aged twenty, has been brought up in the service of children; frequent attacks of both diurnal and nocturnal epilepsy, which have caused intellectual weakening, becoming more and more pronounced. Biting of tongue, aura in the shape of a ball, no signs of hysteria, no anæsthesia, no loss of sensibility to touch or temperature, no disturbance of the muscular sense. This patient has the singular faculty of passively preserving for more than half an hour the positions which are given to his limbs, or which he will take himself, and this he does with his eyes open. In this condition his muscles only show a slight degree of stiffness.

CASE II.—F., aged twenty-six years. Epileptic since the age of nineteen. Slight muscular spasms; possesses the same power as the other patient, but more pronounced; he remains, it seems, longer in the same position. There is no stiffness with him when a limb is extended; besides, there are no indications of hysteria, no loss of feeling or impairment in the muscular sense.

These motor disturbances become graver in melancholia, and especially when associated with stupor. We will return to this important point in the nature of the stupor later on; for the present we will only remind our readers that it is under this name that many authors have described, and still do describe, the katatonia of Kahlbaum.

Guislain[1] also notices these symptoms in the condition which he names *ecstasy;* differing from the ecstasy of others especially of hysterical patients, and which also presents the symptoms we have under review (Morel,[2] Michéa[3]). They may all accompany symptomatic melancholia; one meets them for instance, during the periods of depression in circular insanity (Ritti) and in the different forms of alcoholic intoxication, where they are as it were the rule, occurring as shocks, spasms, or convulsions. We have recently had the opportunity of observing a case of alcoholism in a woman who, suffering from panophobic stupor presented these symptoms under three different forms: muscular spasms, general rigidity and cataleptiform immobility. M. B.

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[1] Guislain, *loc. cit.*

[2] Morel, *Traité clinique des maladies mentales*, 1860, p. 491.

[3] Michéa, *Dict. de Jaccoud.*, Art. *Extase.*

Battaglia[1] has also observed cataleptiform conditions in an individual intoxicated by haschich, and he accepts in this case the hypothesis of primary hysteria.

In the other forms of melancholia which do not present symptoms of depression, but rather those of simple anxiety, we also meet symptoms of the same nature, very different however from the tremblings so often found in the anxious melancholic. One of us has been able to procure a case of this nature, and although the observations are very incomplete, it may be interesting to report it, as it presents several particulars which associate it very closely with those published by Kahlbaum in the memoir which we have analysed.

Case III.—Mrs. C., aged thirty-four, brought under our care on September 1st, has been subject for the past fifteen days to an attack of anxious melancholia, this being her fourth attack. We are unable to give any information regarding her previous illnesses. There is very great anxiety, panophobic terrors, numerous hallucinations of sight and hearing; she sees dreadful animals, hears voices who tell her that she is a murderess, and that she has murdered also her children. She has ideas of her own guilt, thinks she must poison herself, and fears for her relations; is continually sighing, never remains in one position, but is constantly walking about like a drunken woman, swinging her arms, and always about to fall. At other times she is agitated as one in despair. She repeats everything spoken to her, or which she hears around her. Spasms affect the right arm, giving to that limb almost a rhythmic movement comparable to *chorée malleatoire*. We have not discovered any hysterical symptoms.

September 30th.—She is no longer anxious, but depressed, still retaining her melancholic ideas. She works a little. No more movements of the arm.

October 20th.—She has again hallucinations, and is anxious, *continually repeating: "My poor children, my poor children;" walks backwards. There are again jerking movements of the right arm.*

November.—She calls herself a criminal, thinks she is about to be guillotined, that we are going to boil her. Is very anxious, and has the aspect of a maniac, cries, sighs, and is continually in motion. She has the staggering gait of an intoxicated person. She talks and *sings in a pathetic tone all the little incidents of her life*. There are still jerking almost continuous movements of the right arm.

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[1] B. Battaglia, *Sul haschich e sua azione* (*La Psychiatria*, 1887, *Anno* 5 *fasc.* i., p. 21).

This patient, whom we have lost sight of, recovered some weeks later.

These particular symptoms may also be found in hypochondriacal mania (Cullerre) where Morel[1] has already noticed them. They may also exist in addition to the excitement (Lagardelle). We know moreover that in the gravest form of excitement, more particularly in acute delirium, that disturbances of the neuro-motor system can be most serious. Krafft Ebing[2] describes likewise katatonic symptoms in dementia.

Even in general paralysis at some stage or other we may meet with a variety of katatonic symptoms. Alongside of symptoms called paralytic we may meet convulsive attacks, and a condition of rigidity as described by Kahlbaum is found in cases of katatonia. We have recently had the opportunity of observing two female general paralytics at the Salpêtrière in whom the stiffness was so marked as to resemble permanent contraction. In one case especially that of a patient who maintained her limbs in a condition of forced flexion, to prevent permanent deformity and ulceration (which might have been produced by her nails) it was necessary to apply an apparatus fitted to her arms and hands; there were however neither contractions nor retractions. Instances of this kind are very common indeed and we will not further insist upon them. Nevertheless we may here call attention to an interesting work by Knecht,[3] reporting examples of the combination of general paralysis and katatonia. M. Sage[4] has from his standpoint studied choreiform movements in general paralytics.

To sum up, we thus see that katatonic phenomena taken singly have nothing to characterise them, for they are found in a multitude of mental affections. Apart from accidental motor disorders, such as spasms or contractions, which one may meet outside mental diseases properly speaking, there are motor disorders which belong specially to insanity, and which can be present in the most varied forms of mental disease. Morselli[1] divides them into states of increased reflex excitability of the muscles (tetany), increased muscular tonicity (catalepsy), and states of abnormal distribution of central motor impulses (such as stiffness at the beginning of a movement).

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[1] Morel, *Traité des mal. ment.*, p. 712.

[2] Krafft Ebing, *loc. cit.* Bd. i.

[3] Knecht, *Ueber die katatonische Erscheinungen in der Paralyse* (*Allg. Z. f. Psych.*, Bd. xlii, 1886).

[4] Sage, *Thèse de Lyon*, 1884.

Consequently we may say with Arndt,[2] that the insanity of tonicity (*Spannungs-irresein*) is not a disease, but may develop itself upon the most diverse grounds and under the most varied conditions.

Further, considering them only in the cases called katatonia, their mode of development, course and relations with the other symptoms have nothing to specify them and they present no regular characteristics. We may meet them in all the stages and during the whole course of the disease, or only during a limited period; they may be predominant or considerably effaced, and in their relations to the insane delusions they have been spoken of as primary, secondary or independent. Let us add also, that in their essentials even, they seem to us to differ completely from each other. First, their external manifestations present very varied forms; then they can be spontaneous or not, and we readily admit that if they are mostly the consequence of insane ideas there is nothing impossible in the fact that their direction may be changed, or at other times new symptoms may be suggested. On the other hand they may be independent of any insane idea, and we can see that with certain patients they appear to be subject to the influence of the will and are only a simple phenomenon of attention produced apart from all indications of effort, or at least with a minimum of effort, disappearing when the attention of the patient is fixed or directed to some other point. This fact has been ascertained in one of M. Ch. Féré's patients, and in the case of a woman whom we observed at Salpêtrière. In other cases these phenomena seem to be outside the domain of the will, independent of the patient's attention, and performed unconsciously; admitting however—as we stated in our last observation or case—of explanation by referring to alterations of the muscular sense.

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[1] Morselli, *Manuale di Semejotica delle mal. ment.*, Turin, 1886.

[2] Arndt, *loc. cit.*

B.—Alongside of these symptoms there are others in closer relation to the psychical disturbances, and which Kahlbaum states are as equally characteristic of katatonia. He even considers them from the pathogenic point of view as being of the same nature as the preceding ones. Let us cite as examples, verbigeration, dumbness, stereotyped gestures, pathetic attitudes and systematised resistance. It is thus that, dominated by the idea that the spasm-element occupies a leading place in the disease he describes, Kahlbaum ascribes the verbigeration to a spasm of the speech muscles, due to an impulse from the central speech centres; the same might occur in the case of dumbness which might be due to a tonic convulsion, as opposed to verbigeration, due to a clonic one.

Similarly also, the stereotyped gestures might be ascribed to acts of combined spasms. It will be sufficient for us merely to signalise these statements; such physio-pathological hypotheses cannot even be discussed. They may be ingenious views of the mind but they do not form a foundation solid enough to build a new nosological form upon. Moreover, whatever may be the explanation of these phenomena, considering them from the purely clinical point of view, they have not in our opinion the importance which Kahlbaum gives them. We note in the first place that after having given the verbigeration as an important diagnostic sign, Kahlbaum adds that in the course of the disease it may transform itself into any of the other forms, from which he had previously distinguished it (ordinary *Redesucht*, loquacity of the mentally enfeebled, confused ideation, and confabulation). On the other hand, we are of opinion that this verbigeration has nothing that characterises it, for we may meet it elsewhere particularly in cases of primary or secondary intellectual enfeeblement. Vogelsang and Jastrowitz[1] notice it also in general paralysis. Stereotyped gestures comparable to nervous twitchings, are also frequent in the same cases of mental weakening,[2] with or without the co-existence of mania which when it exists explain their nature frequently without the necessity of invoking the theory of coordinated spasms from certain cerebral centres. A well-known instance is that of the sigher (gemisseur) of Morel.[3] As regards the pathetic, strange and sometimes cabalistic attitudes, they exist most frequently in delusional insanity, especially when it is grafted on a degenerative foundation. Indeed, some authors maintain that they are characteristic of this type.[4] The obstinacy and systematised resistance present nothing worthy of remark, for they are mostly to be observed as associated with all cases of the melancholic state; so much so is this the case, that one author has given the name of "the insanity of opposition" to melancholia.[5] Dumbness is also met with in the same conditions, especially in profound melancholia, of which it is almost a necessary symptom, and without there being any coincidence with the least katatonic phenomena.

C.—Neither does the course, called cyclic, of the disease offer anything characteristic; for the variable conditions through which the disease passes have nothing regular in their mode of appearing or in their relative positions. We may convince ourselves of this by reading Kahlbaum's descriptions and observations. This author who with the desire of noting a recurrence of the same phenomena goes the length of seeking in the previous life of the patient some attack of mania that may have happened years before, in order to establish his theory that a maniacal period exists always before the characteristic katatonic attack. He does not take into account the fact that katatonia does not always commence by a maniacal outburst. Not only does this initiatory period of maniacal excitement appear to us to be often one of melancholic anxiety, but many cases present themselves as depressed melancholics from the very beginning of the disease. Considered from another aspect the cycle of the malady presents nothing truly regular. We may easily note more or less regular alternations of excitement, and these attacks present themselves as often under the form of maniacal excitement as of *melancholic anxiety* (Hammond[1]), and further, this depression may increase even to the stage of stupor. Taken in its entirety this would be the general course in successive phases of the forms of insanity noted by Guislain, Zeller and Griesinger. Let us add also that Kahlbaum recognises "that mental diseases in general, including katatonia, begin with melancholia, pass into mania, next into *Verwirrtheit*, and finally end in dementia." In another place he says: "Melancholia attonita which has been considered until now as a special form of disease *develops itself primarily in very rare cases;* it pursues in general rather a course of simple melancholia, or a condition of melancholia following mania in such a manner that the melancholia attonita is the third phase of the complete process which terminates in recovery or dementia." This is an incontestible clinical fact which has moreover been known for long, and frequently verified (Morel,[2] Guislain, Griesinger,[3] Dagonet[4]), so much so that "for the same attack of disease there are four constituting phases. It follows from this according to Guislain, Zeller, and Griesinger, that the various forms of insanity have different phases, and that, no more than simple melancholia mania or dementia, can melancholia attonita either be considered to be a particular form." This argument is perhaps excellent for Kahlbaum who wishes to believe that simple melancholia attonita is merely a mild form of katatonia. But for us who admit with difficulty a katatonia without katatonic symptoms, and who hope not to be contradicted when saying that many cases observed in asylums do not present the katatonic symptoms of Kahlbaum—for us therefore the above argument seems to have little weight; and as we have just seen that he also associates the course of katatonia with that of other forms of insanity in general, we say of it what he says of melancholia attonita, namely, that it must not be considered as a particular form, at least as judged by its course. We have likewise seen what can be said in support of its symptoms in particular.

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[1] *Allg. Z. f. Psych.*, Bd. xlii., 886, p. 881.

[2] R. Brugia e S. Marzocchi, *Dei movimenti sistem. in alc. forme d'indebo. ment* (*Arch. ital. per le mal. nerv.*, September 1887).

[3] Morel, *Etudes cliniques*, vol. i., and *Traité des mal. ment.*, p. 713.

[4] Tanzi and Riva, *La Paranoia, contrib. al. storia delle degeneraz. psichiche* (*Riv. sper. di fren.*, 1884-5-6).

[5] Guislain, *loc. cit.*

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[1] Hammond, *loc. cit.*

[2] Morel, *Traité des mal. ment.*, p. 489; and *Etudes cliniques*, vol. ii., p. 257.

[3] Griesinger, *loc. cit.*, p. 295.

[4] Dagonet, *loc. cit.*

To sum up, isolated, not one of the symptoms which we have passed in review can by itself characterise a special psychopathic form of disease. Is it otherwise with them when considered *in toto?*

IV.

In short, in order that a union of symptoms not characteristic in themselves may constitute an essential pathological entirety, it is necessary that they possess among themselves close relations with regard to their nature, origin, mode of succession and causation, in such a manner that notwithstanding their inevitable variations, one can always grasp their relations, recognise their connections, and refer them to a defined primitive type, and to a common superior cause.

Now this is not the case here; we do see a co-existence in the description of katatonia, but not an association or a combination of symptoms. Whatever the advocates of katatonia may say of it, the difference between it and general paralysis is great. The origin of the latter rests on a solid foundation of anatomical lesions, the intimate nature of which is perhaps not yet absolutely known, but which is sufficient even with our present imperfect knowledge to create a morbid class, and perhaps in time we may succeed in determining its varieties. Here on the contrary nothing is precise, for the meningitic lesions of a tubercular nature found in some cases are not the anatomical characteristics of the disease; and they all rest on hypothetical data of hyperæmia (Meynert) producing vaso-motor contractions, or spasms from certain cerebral regions (Kahlbaum, Neisser) which are variable, multiple, and which we can only vaguely determine owing to the imperfection of our actual data as regards the anatomy and physiology of the brain.

Kahlbaum admits himself that the pathological anatomy has yet to be made and Kiernan after long dissertations arrives at the conclusion that the essential characteristic is a primary vaso-motor dilatation; leaving aside the meningitis, which one might imagine he should have utilised for the purpose of making a suitable anatomo-pathological foundation. Besides, as we have seen, Brosius divides katatonia into three groups, which resemble one another little anatomically. Other authorities do not refer at all to the pathological anatomy. Therefore the anatomical lesions are absent, and as nothing does authorise us to associate these phenomena (which we may suppose to be of dynamic order) to identical known symptoms resulting from an organic cause, it follows that the pathological structure cannot show a solid or rational foundation.

But in the absence of an anatomical substratum and determined physiological data by what can a morbid form characterise itself? We are left to deal with the symptomatic evolution and the ætiology. Again, the absence of a certain anatomical criterion necessitates extreme care in the choice and classification of the phenomena and of described examples of the condition. Now even the classification of the varieties of katatonia which Kahlbaum gives at the end of his memoir show us, even if the reading of the symptomatology and his observations had not already convinced us, how varied were their intensity, mode of appearing, succession, and even the evolution of the different stages of the disease. It seems to us superfluous to dwell upon this point further, as we have already exposed these fallacies in the early parts of this work. We should only be repeating ourselves.

As regards the ætiology, which if we exclude certain particular exceptions fails to assist us in making a differential diagnosis in general medicine, the causes above recorded are absolutely insufficient in cases of mental diseases, where ætiology is one of the most unsolvable problems to which the alienist physician devotes his energies daily. One may know the very numerous causes of mental disease in general; but it is almost impossible to determine the action of a particular cause in a given case, when considered from the point of view of its origin, or the particular form it may take, and the special course that it may follow.

Moreover, the ætiological causes which Kahlbaum gives are perfectly common-place ones, and such as we may find at the source of all possible forms of mental disease. There are however two causes which in our opinion might induce a special predisposition and serve to characterise the foundation on which the disease develops itself. These are: degeneration in general, and the hysterical state. These two factors are not mentioned by its advocates, and yet they seem to us to be of great importance. In fact, we find in the description of katatonia, most of the features common to all hereditary forms of insanity; such as an alternation of the delirium with the succession of exalted or depressed states (Morel[1]), monotonous verbosity, incoherent or emphatic language, theoretical poses, strange cabalistic attitudes, a predominance of poetical, theoretical, or mystic ideas, such as are described in Kahlbaum's observations, and so frequently noticed as to induce Schule to consider katatonia as a kind of religious Wahnsinn. Let us also call attention to the frequency with which psychical disturbances occur at certain biological epochs in those with a hereditary tendency to mental disease; we will then not be surprised that certain authors should have classified katatonia amongst the insanities of puberty (Maudsley[2]). Besides, most of the causes noted in the observations of katatonia do commonly act only as occasional factors on individuals having more or less a hereditary taint. Let us here cite as examples sexual excesses, onanism, puberty, accouchement, menstruation and alcoholism, upon which Kiernan insists. Finally, our study of the observations shows us that even the majority of the patients present different characteristic features, indicative of mental degeneration, and sometimes even of physical deformities, the importance of which has seemed to have escaped the observers' notice, who we must say have in the examination of their patients paid insufficient attention to the question of degeneration in general.

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[1] Morel, *loc cit.*, p. 479.

[2] Maudsley, *loc. cit.*

With regard to hysteria which especially in its maniacal forms may approach and even be classed with degenerative states, it partakes of the same characters as above enumerated, but it may also favour the production of katatonic phenomena properly speaking. Is it not in hysteria chiefly that one observes more or less defined convulsive seizures of a hystero-epileptic character,[1] attacks of catalepsy, lethargy, muscular contractions, ecstasy, and choreiform movements, without taking into account simple hyper-excitability (neuro- and cutaneo-muscular) and muscular flaccidity while awake? Now hysteria has never been seriously sought for in the observations of katatonia which we have read of; only very briefly and in a vague manner have researches on sensitive anæsthesia been referred to. The nature of the attacks is always badly defined and in very general terms; they are called hysteriform or epileptiform seizures, &c., or perhaps one is satisfied with saying that a patient has a hysterical aspect. Yet we would be readily inclined to think that a more attentive research could have disclosed in the patient the presence of some hysterical symptoms, and this so much the more because we meet in reading the observations symptoms which are often enough found in hysteria to induce one to be arrested by them. However, their importance does not seem to have been understood, or they have been associated with the katatonia, whereas in some cases they have preceded it by several years. We also find tonic spasms occurring in episthotonos in the course of convulsive seizures, re-appearing several times in one day (Case II. of Kahlbaum); uncertain convulsive attacks (as found in almost all the observations) and followed later by involuntary attacks of weeping and laughter (Case III.); loss of sensibility of the pharyngeal mucous membrane (Case IV.); recurring attacks of laughter at the same hour for several years in a nervous subject (Case I. of Hecker) before the beginning of the katatonia which was moreover followed by hebephrenia; convulsive attacks associated with episthotonos, with intense delirium towards the termination of an attack of typhoid fever and before the first symptoms of katatonia appear (*Ibid,* Case II.); a similar attack presenting some of the characters of *the arc de cercle* (Case IV. of Neisser), occurring in a woman in whom Krœpelin had suspected the existence of hysteria, but this factor had been neglected by Neisser (Obs. IX.). In two other cases referred to by the same author we find somnambulism, with black visual hallucinations (Case X.), prolonged cough without pulmonary phenomena, attacks of syncope, and repeated seizures of vomiting and cephalalgia and amyosthénie (Case XII.). A fact which seems to confirm our conception of viewing this condition, is the peculiarity noted by Kahlbaum, of convulsive epidemics of katatonia. It would be also interesting to ascertain the hysteric influence in these cases especially when the disease sets in suddenly by stupor after an accident; this may be only a hypothesis, but it presents at least nothing improbable, since the more recent researches of the School of Salpêtrière have brought to light the great importance of traumatism in developing morbid manifestations when acting on a hysterical basis. This neglect of attention to hysteria which we have noted is no doubt due to the fact that the majority of German physicians seem to take little notice of hysteria in general and oppose determinedly the theory advocated by M. Charcot, that male hysteria is of common occurrence.

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[1] Morel (*Études Cliniques*, vol. ii., p. 285, and following) reports under the name of "stupidity" cases which seem to us analogous to katatonia, and he rightly considers the presence of the principal phenomena (called later on katatonic), and the special course of the affection to be connected with a state of mental degeneration, and of very grave prognosis. Elsewhere (*Traité des mal. ment.*, 451), he says that the phenomena of ecstasy and catalepsy are associated in a more intimate manner with the history of epidemic insanities, especially to that of religious mania, and to certain neuropathic conditions, such as hysteria.

Schule is the only author who has occupied himself with a study of these neglected factors in the examination of his patients, and his observations have finally induced him to describe a form of hysterical katatonia (sixth type of *hysterischer Wahnsinn*), and from another aspect he classifies katatonia in general among the psychoses developing themselves in a morbidly modified (*invalide*) brain.

Besides, the study of hysteria in this class of patients may be interesting to explain, at least in certain cases, the production of some of the motor disturbances. We know how frequently the muscular functions become altered in hysterical patients and it is not irrational to suppose that a connection might be found to exist between those alterations and the particular spontaneous or induced attitudes assumed by the subjects of katatonia.

We here report the case of a hysterical patient whose symptoms presented many features of resemblance to that of the katatonia of Kahlbaum, and in whom we discovered slight disturbances of the motor functions.

CASE IV.—Mdlle. L. Ch——, aged twenty-four, admitted to Salpêtrière on June 24th, 1887.

Hereditary Antecedents.—Father, weak, both mentally and in character. *Mother*, nervous, but without (hysterical) attacks; of feeble intelligence; had tremblings of the head. No consanguinity. No definite information regarding the grandparents.

Personal antecedents.—Nothing particular noticeable in childhood. When thirteen years of age Mdlle. Ch—— seems to have become subject to illness for the first time. She was then a little nervous, and was treated for anæmia. At the age of twenty a convulsive attack of hysterical character is first recorded; then a second attack three or four months afterwards; and after that these attacks returned about every month; never by night. At the same time she had slighter incomplete attacks, with giddiness. She suffered also from frequent headaches. In disposition she was willing although rather weak, and she was always a "spoilt child." Two years ago she became associated with a young girl who occupied herself with spiritualism, and who rapidly gained a great ascendency over her. That girl soon persuaded Mdlle. Ch—— that the latter could be cured by her through magnetism, and they commenced to practise without the knowledge of her parents. The attacks, however, continued, the last occurring on May 19th. She last menstruated on June 6th.

The delirious symptoms date from thirteen days ago. She began to show signs of agitation; was no longer mistress of herself, of her thoughts and actions; visual hallucinations have existed since the first day; there is insomnia, but no *premonitoir* dreams. On the third day hallucinations of hearing occurred; her friend tells her that she magnetises her. She answers that her friend has deceived her by not curing her. Her agitation increases and she makes passes as if she were magnetising somebody. She even tries to magnetise herself so as to cure herself of a condition similar to her friend's, who is infirm in one leg, and has been magnetised for it without result. She becomes loquacious, speaking often in her delirium of a letter which in her practices of spiritualism, her friend had caused to be written by her brother, who died twelve years ago. Since that occasion her agitation has steadily increased; refuses food; absolute sleeplessness.

June 24th.—Actual condition. Violent maniacal agitation. We are able however to fix her attention for a moment at a time by insisting or shaking her energetically. She is dishevelled, hardly dressed, feet naked, has hallucinations. There is excessive facial mobility; her actions are disordered. She expresses incoherent language, all her words relating seemingly to magnetism. She cries, makes incantations, and assumes cabalistic gestures; she arranges the chairs around her, and seems to magnetise imaginary persons who are supposed to be seated thereon.

There is no evident sensorial anæsthesia. An examination of the sight is very difficult; however she names colours correctly, and there does not seem to be any alteration in the visual field. Both iliac fossæ and the lower mammary regions are painful on pressure. There is facial asymmetry, the right side being the smaller. The nose deviates to the right side, the right eyebrow is on a lower level than the left and there is discolouration of the left eyelashes, dating from infancy. The palate is malformed; the ears, hands, &c., are well proportioned. The skull is regular and symmetrical.

Maximum transverse diameter	13.9
Antero-posterior diameter	17.9
Bizygomatic diameter	12.1
From between the eyebrows to alveolars	6.5
Maximum horizontal circumference	53.5
Anterior half circumference	25.5
Posterior half circumference	28
Transverse curve (from one tragus to the other) ...	32
Antero-posterior curve	34

July 2nd.—The agitation continues; the hallucinations are at all times very numerous. In the day time she lies down on a grass plot in the garden and remains during some ten minutes with her eyes shut, and her neck and limbs stiff, as if in a condition of contraction and declines to respond to any external impressions.

July 3rd.—She begins to have confused ideas about her left hand and asks herself whether it can at all belong to her. All at once she extends her right arm and remains thus several minutes fixed and immovable without answering the questions addressed to her.

July 6th.—Very excited, but chiefly anxious, face restless, dishevelled, weeps and laments much. Hallucinations (of sight and hearing) are at all times very numerous, intense and almost continual; however, by insisting, one can fix her attention. At times she assumes attitudes resembling the passionate position of hysteria, and the few phrases she pronounces are uttered in a reciting pathetic tone. She continually repeats that she is dead, and no more herself. Her limbs remain for some minutes in any position we may place them in, or which she has assumed herself, but are not rigid—a cataleptoid condition. Loss of control of the rectum and bladder (gâtisme).

July 8th.—Is much calmer, but the hallucinations continue although she says they are not so numerous. Her sentences are unconnected, apparently in reply to her hallucinations. There is an induced cataleptiform condition for some minutes at a time, the right arm being extended, and the left arm in a semi-flexed state. Her face reveals no fatigue, respiration normal, the arms retain absolutely their position, following only the movements of the whole body. Slight trembling of the extremities, with a flexibility of portions of the limbs, which retain the various positions given to them successively.

Muscular sense of left arm somewhat disturbed. On this side she is unable to find her hand if her eyes are shut. The estimation of different weights is less accurate on the left arm than on the right. Contact and pressure are not so well perceived on the left side as on the right, and pricking seems to be less distinctly felt on the right side.

She continually repeats that she is dead and without a body; that there only remains to her a left leg, a right arm, and two eyes; that she is "retournée," and to take her right hand with the left one she passes the latter behind her back, because she is "retournée." When one fixes her attention this disappears momentarily, otherwise she is very anxious about it. She weeps, is disconsolate, says that everything is changed around her, that she no longer has a personality, and yet she is indeed L. She believes herself persecuted by another patient (female); she has caught the disease of another, and this has caused her left leg to become immovable. The parts of her body not belonging to her have been attached to her, she doesn't know why. She calls out for her parents; generally she is gentle and calm; at times she has attacks of anxiety. In the afternoon she remained for a long time lying extended on the grass, her arms being extended crosswise; she tells us that this is a conjuration.

July 9th.—The symptoms vary very much however; she is no longer maniacal as on admission. She assumes "theatrical attitudes," especially when agitated, and when speaking again of magnetism. Her ideas run always in the same groove; she repeats them continually, but never uses the same words; she has no special vocabulary. She assumes peculiar attitudes, holding her hands crossed on her knees; the right hand on the left knee, and *vice versâ*. In the morning she calls herself complete, and recognises her left hand by a spot of smallpox she has on the forefinger; however, some moments afterwards she recurs to the ideas of the previous evening, and she even says she has lost both her eyes. She is unable to explain herself because she is perplexed.

July 16th.—She assumes cabalistic attitudes, crosses her legs when holding herself upright, to prevent misfortunes; keeping her knees as above noted. She remains fixed and immovable in these positions. These induced cataleptiform states continue for some minutes. She has been very calm, and somewhat depressed for some time; speaks little, isolates herself, remaining apart from the other patients, weeps much, is still sleepless, and always has loss of control of the rectum.

August 1st.—There is considerable improvement, no more excitement, no attacks of sadness or tears; she asks for her family and worries herself as to what will become of her; she thinks her illness may do her harm in the future, or return, &c. There are no more cataleptic attacks and she is beginning to sleep.

August 16th.—Is no more delirious, sleeps and works well and may be considered as cured. Physical examination shows nothing noteworthy in particular; no signs of hysteria.

September.—Menstrual functions re-established. Recovery completed. Discharged.

The following case, which one of us had been able to study at a previous time, presents likewise many of the features given by Kahlbaum as characteristic of katatonia, and here again we find both heredity and hysteria well proved.

CASE V.—Mdlle. N., aged nineteen years.

Hereditary antecedents.—The grandmother of the father of the patient died demented, as also her son (the patient's paternal grandfather). The father himself is a very nervous man, strange, violent, and subject to arthritis.

Personal antecedents.—Little is known regarding her childhood; she has always been very nervous, strange and whimsical. For some years she has been suffering from and treated for hysterical symptoms. In January, 1884, she became sad without reason and courted loneliness; in June, she had an attack of maniacal excitement, becoming insubordinate, wilful, attempting continually to escape from home; fancied that the Shah of Persia loved her, and was going to ask her hand in marriage, and she was continually singing comic operatic airs. In July this exctiement was replaced by melancholic depression; she then feared that she was going to become sick; that she had cholera and was going to die, begging pardon from everybody for her errors, as she stated a voice commanded her to do so, and she believed that the worms gnawed at her. At this time she presented a state of rigidity almost general but without contraction, one having some difficulty in overcoming the resistance of the muscles, and when the limbs were moved they became immovable in the new position given them, but remained rigid.[1] This condition of melancholia became more serious, and gradually merged into a state of stupor with dumbness, refusal of food and progressive emaciation.

August 22nd.—Actual condition. She has the expression of melancholic stupor—fears, absolute dumbness, complete refusal of food, extreme emaciation, she cannot even sit or stand, but lets herself fall about as if she were an inert mass; breath fœtid, urine scanty, and constipation. There is no lividity or œdema of the extremities, and she suffers from amenorrhœa and sleeplessness. There is almost complete analgesia; no certain signs of hysteria, and no stiffness. In stature she is tall, of regular conformation, head small, face asymmetrical, teeth irregular and the maxillary bones much incurved.

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[1] For this information we are indebted to the kindness of M. Ch. Féré Physician of Bicêtre, who saw the patient at that time.

Treatment.—Extra nourishment by the tube (meat powder, soups, broth, milk, Peruvian quinine, bark wine, mixed with Bordeaux wine and arseniate of soda), mustard baths, and syrup. morphiæ.

August 26*th.*—There is an improvement in the general condition, dumbness, and refusal of food. She now resists the tube, which she had at first passively accepted.

August 27*th.*—She says: "I have typhoid fever; let no one come into my room."

August 28*th.*—Her expression has improved; no alteration in the mental condition; dumbness; refusal of food; systematic resistance to everything she is asked to do; but there are no motor disturbances, and she walks a little alone. She weeps much.

August 30*th.*—In the same condition; sensation is much better, although it is always a little dull, without special localisation.

September 1*st.*—She eats alone, the dumbness continues. Hydro-therapeutics.

September 11*th.*—She says, "I can't sleep in this bed, for they say it has been *offensé.*"

November.—Towards the end of this month she menstruated. She still remains in the same condition; speaks only very softly and when alone, and if one speaks to her, she laughs and cries at the same time, but does not answer.

December.—Again she refuses her food on account of hallucinations of hearing; she seems also to have visual hallucinations. She speaks a little, and asks to be allowed to go away, because she costs so much, and that her father will be ruined thereby. Menstruation regular. On the 22nd of this month she wrote, "It is expected that I will be found dying from one moment to another; one desires to kill me in consequence of mad ideas which concentrate themselves in me; I am in such a stupid state that I don't know what I do; I lose my reason; I can speak no more; I am unable to do anything; I do not deserve to live; everybody finds me stupid, and this is true; I lose my reason." At the same time she made figures, which she placed in a peculiar order; this is an exact copy of them.

1 k. 250
1 k. — 1000
250

384579
123456

261123

$$\frac{3}{8} \quad \frac{7}{9}$$

$$\frac{3 \times 9}{8 \times 9} \quad \frac{27}{72}$$

1000 — k.
250

123456
384579

$$\frac{7 \times 3}{9 \times 8} = \frac{21}{72}$$

(*Vide* Note 1.)

January 15*th*, 1886.—She eats a little, but only bread, which she buys by measure; she works a little and talks well; it is the Lord who speaks to her, and has forbidden her to eat, to expiate her sins. God told her to eat nothing; that she must die for she was not worthy to live. When she laughed, that was because voices spoke to her. She does not confess to hallucinations of sight, and believes firmly in the reality of God's voice.

January 20*th*.—She is no more delusional; is a little excited and insubordinate, crying without cause, but it relieves her; she occupies herself usefully but in a feverish manner. She is conscious of her past condition, and relates the delusions she had about her guilt; that she believed herself to have been the cause of the death of her mother, and that the Shah of Persia loved her. She heard the voice of God speaking to her in an imperative tone. She says that all these symptoms have entirely disappeared. There is still some genital excitement, inveterate self-abuse. These practices date, she says, from a very long time ago, and had been taught her by one of her female relatives, whose bed she shared when a child. There are no signs of hysteria. Discharged recovered.

November 15*th*, 1886.—The delusions have not appeared again, but it is possible to observe in her the presence of the *essentials of hysteria*, and symptoms of the same nature, analogous to those noted before the appearance of the delirous troubles.

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[1] We might readily compare this with the copies of writings given by Neisser, in which the same words or symbols are repeated in such a manner as to constitute a kind of written verbigeration.

V.

We have finally seen what katatonia is, and we have explained the conclusions which have seemed to us to result from the examination of the descriptions of this disease, and of the observations given in its support. Kahlbaum's proposition has on the whole as yet been accepted by few writers. Having considered its advocates, we note certain differences existing in their writings, and more especially did we refer to Schule, whose description of the disease is in our opinion much nearer the clinical reality than any of the others.

To sum up, in order to create katatonia, Kahlbaum insists on these two points: (1) The non-existence of attonity, except as a symptom, and (2) the circumstance, that the essentially katatonic phenomena becomes the characteristic features of those cases in which attonity is present.

One may dispute the entity of the stupor, and this opinion has nothing extraordinary in it. Everybody admits the possibility of stupor in any mental disease, but some authorities go no further than that; others however describe a special form of stupor, and amongst the latter, some consider it as a distinct disease, others, a more numerous class, connect it with melancholia. But, because in this last case the malady could follow the course indicated by Kahlbaum—passing through a stage of simple melancholia, or even through one of anterior mental exaltation (mania or anxiety), must it therefore be concluded that the stupor ought to be entirely rejected under the pretext that it is only a phase of the disease when considered in its entirety? This seems to us most irrational, for it is the stupor which constitutes in those cases the critical stage of the disease, the other phases being only premonitory, and often of short duration in comparison to the period of stupor. Let us add that the latter condition may exist as it were from the beginning of the disease. Because a malady cannot constitute itself at once from all its symptoms, and may pass through different stages before arriving at the critical state, must it therefore be rejected? If this were so, then there would be very little left of mental pathology, for there are no mental affections which form themselves *de novo*, or remain identical during their whole course; and is not mental exaltation, and more especially depression or moral hypochondria, at the beginning of all forms of mental disease? We have further seen that Kahlbaum admits this himself, and to be logical, if we accept his argument in regard to stupor we must extend its application to all the other forms of psychical derangement.

The second point on which Kahlbaum insists is the necessity of giving the priority to the katatonic symptoms, which may be more or less pronounced, but the existence of which should form the rule in all cases of melancholia with stupor. We have already remarked that this seems to us a singular exaggeration. Very few of the published cases of melancholia with stupor present any symptoms called katatonic. We have seen several such cases, even after our attention was directed to this subject, and there is perhaps no alienist who has not observed similar ones. It is certain that if dumbness and opposition are to be considered katatonic symptoms, then all cases of pure stupidity, as well as many melancholics, are katatonics. But we have already given our opinion on these psycho-physiological exaggerations. We have also seen that one might express the same unfavourable criticism on these phenomena as Kahlbaum applies to stupor (atonity), *i.e.*, that they are only symptoms presenting themselves in nearly all psychopathic forms, and that when they show themselves as prominent features and as associated with the conditions called melancholia attonita they do not constitute a regular entity either in their course, or in their mode of arrangement, or even in their intrinsic characters.

We may complete this study by saying, that Kahlbaum's attempt does not seem to us so far sufficiently justified. We might repeat in substance with regard to katatonia what M. J. Falret[1] said before concerning catalepsy, namely, that in the description of this affection, some authors have coupled together facts which, from different points of view, are dissimilar; and that they have rather recorded the history of a symptom (or better, of a "syndrôme"), than of a veritable disease.

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[1] J. Falret, *De la catalepsie* (*Arch. gén. de Méd.*, Aug., 1857).

If we consider further that from the physical point of view the prominent symptom is the presence of disturbance of the neuro-motor functions, whilst the principal psychical feature is a more or less acute condition of melancholia (the other symptoms, progress, &c., presenting nothing special), we are certainly of opinion that for the present katatonia must be classed under the general group of stupors—simple or symptomatic—of which it may only be a variety more closely connected with a degenerative and more particularly hysteric ground.[1] We must add, that this conclusion is not an explanation, but we think it to be the only opinion which can be formulated in the present state of science. We will leave to other writers, more competent and bold, the chance of venturing upon the path, still very imperfectly known, which leads to the elucidation of the various forms assumed by the hysterical psychoses, and to define, if possible, the domain, so extensive and so vague, of mental degenerations.[2]

Since the publication of this paper in the *Archives de Neurologie*, Dr. Hack Tuke mentioned a communication made to him ("Mental Stupor," Transactions of the Int. Méd. Congress, London, vol. III., p. 634). He does not agree with Kahlbaum, but thinks that the cataleptic phenomena are due to a mental state of absorption under the influence of a sad hallucination. He separates sharply "mental stupor" from acute dementia, which has often been confounded with it.

Drs. Séglas and Bezançon (Nouvelle Iconographie de la

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[1] In these cases, when we are able to discover actual or retrospective delusional ideas, they are mostly of a mystic nature, as has been remarked by Morel, Schüle, &c.

[2] The reader may also consult on this subject, Lanfenauer, *Ueber Katatonische Verrücktheit. Orvosi Hetilop*, 1882. Konrad, *Beitrage zur Lehre der Katatonie, Orvosi Hetilop*, 1882. Dunkerlost, *Ueber Ætiologie und Behandlung der Katatonie, Ned. Ver. f. Psych.*, 1888. Hammond, 'Treatise on Insanity,' London, 1888. Spitzka, 'Insanity,' New York, 1888. Edward Geoghegan, 'Case of Prolonged Maintenance of a Fixed Position' (*Journal of Mental Science*), 1882.

Salpêtrière, March and April, 1889) relate a case of cataleptic melancholia, presenting a cyclical progress with the peculiar symptoms attributed by Kahlbaum to katatonia. This paper contains pneumographic tracings and myographic tracings upon patients during her cataleptic attitudes, proving that they are accompanied with no effort. Moreover the patient, as those mentioned in the present review, bore evidence of other hysterical alterations.

CEREBRAL LOCALISATION IN ITS PRACTICAL RELATIONS.[1]

BY CHARLES K. MILLS, M.D.

FROM the clinical and pathological observations of practical physical physicians sprang the great conceptions out of which have developed the science and art of localisation. Gall,[2] from outward form and on uncertain grounds, located speech above the orbits; in 1825 its pathology and morbid anatomy were first clearly indicated by Bouillaud,[3] who held that in the anterior lobes of the brain resided the organ of speech; and Broca,[4] in 1861, from pathological observations, definitely placed the seat of articulate language in the gyre which bears his name. In 1864, J. Hughlings Jackson[5] suggested that certain convolutions superintended those delicate movements of the hands which are under the immediate control of the mind; and an observation of Hitzig,[6] that certain ocular movements and other muscular phenomena occurred during the galvanisation of the heads of patients, led in 1870 to those researches with Fritsch, which have immortalised the names of both.[7]

The researches of S. Weir Mitchell[8] on the physiology of the cerebellum constituted an early and important contribution to encephalic localisation. From numerous physiological experiments, chiefly on pigeons, both by methods of ablation and of chilling or freezing, he concluded that the cerebellum was a great reinforcing organ, capable of being more or less used in volitional muscular motion; but while believing this he was not prepared to assume that it had no other function.

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[1] Paper read before the Congress of American Physicians and Surgeons, Washington, D. C., September 19th, 1888.

[2] Gall et Spurzheim, *Anatomie et physiologie du système nerveux.* Vols. i.-iv. Paris, 1810-1819.

[3] *Traité Clinique et Physiologique de l'Encéphalite*, p. 284.

[4] *Bull. de la Soc. anat.*, T. vi., Août, 1861.

[5] 'London Hospital Reports,' vol. i., p. 459, 1864, and 'Clinical and Physiological Researches on the Nervous System.'

[6] '*Untersuchungen über das Gehirn.*'

[7] *Ueber die elektrische Erregbarkeit des Grosshirns.* Reichert and Du Bois-Reymond's Archiv., 1870, No. 8.

[8] *Am. Jour. Med. Sci.*, n. s., vol. lvii., 1869, p. 836.

In 1874, a committee of the New York Society of Neurology and Electrology, as the result of carefully recorded experiments, reported conclusions largely confirmatory of those announced by Hitzig. The committee tested also the effects of excitation of the dura mater.[1]

Dr. J. J. Putnam[2] of Boston in 1874, experimented with faradic currents on the cerebral cortex and the parts immediately beneath. He first found the centres for definite and nearly or quite uncomplicated movements, and the minimal current strength that was necessary to produce these movements, after which, with a sharp knife he made a cut underneath these centres, leaving a good-sized but thin flap which contained these supposititious centres. Having done this, he found if he irritated as before, leaving the flap *in situ*, the movements did not occur. Turning the flap up, however, a slightly increased current strength produced the same muscular contractions. When the flap was turned back and adjusted, and the electrode applied on its surface as at first, the contractions were not produced. Three dogs were used in the experiments, which were made by Dr. Putnam at the Physiological Laboratory of the Harvard Medical College with the assistance of Prof. H. P. Bowditch and Dr. William James. After they were made it came to Dr. Putnam's notice that from the same methods the same results had been obtained in the same year by another observer, Braun.[3] Dr. J. Burdon Sanderson[4] also, in 1874, had announced the same fact.

The first reported physiological experiments on the human brain were those of Bartholow[5] in 1874, who, using both a galvanic and a primary faradic current, passed insulated needle electrodes into the brain of a patient.

In any historical reference to American work, the labours of Wood[6] and Ott[7] on thermic phenomena must hold a high place:

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[1] *New York Med. Journ.*, 1875, xxi., 225-240.

[2] *Boston M. & S. J.*, 1874, xci., 49-52. *Ibid.* 1879, c., 260-262.

[3] *Eckhard's Beiträge zur Anatomie und Physiologie*, vii. 2. Also: *Centralblatt*, Berlin, June 13th, 1874.

[4] Proc. Royal Soc., June, 1874.

[5] *Amer. Jour. Med. Sc.*, Philadelphia, 1874, n. s., lxvii. 305-318.

[6] Fever: A Study in Morbid and Normal Physiology. Smithsonian Contributions to Knowledge. November, 1880.

[7] *Jour. Nerv. and Ment. Dis.*, April, 1884. *Phil. Med. News*, July, 1885. *Jour. Nerv. and Ment. Dis.*, vol. xiv., No. 3, March 1887. *Ibid.* No. 7, July, 1876, p. 428. *Ibid.*, vol. xiii., No. 2, February, 1888.

In 1884 Starr,[1] in a review of American medical literature for twenty-five years before, found records of nearly 500 cases of local disease of the brain, some of great value; such records have since increased and multiplied and what is better, have improved in method and accuracy. The numerous contributions of Seguin here rank first. A brain tumour was removed by Hirschfelder and Morse[2] of San Francisco, February 15, 1886, the fifth case of such operation. Of 63 cases of intracranial operations tabulated by Dr. Park, 17 have been reported by American neurologists and surgeons.

The surgical aspect of cerebral localisation is naturally that which appeals to all as the most practical. In this field unprecedented therapeutic results have been achieved, the crowning triumph being the relief of that most agonising of human diseases, tumour of the brain.

Fascinated by these achievements, we incline to pass by the results elsewhere wrought—in psychological medicine and medical jurisprudence, in general symptomatology and diagnosis, in medical therapeutics and technique. I may however be allowed to devote to these a few fleeting words.

Cerebral Localisation and Insanity.

Bevan Lewis[3] in 1883 pointed out some of the directions in which studies in cerebral localisation might advance our knowledge of insanity, but to those I can scarcely more than allude. He held that the localisation of cerebral function was the outcome of the great principle of evolution carried to its logical issues; that the alienist should rivet his attention upon the changes undergone by the material substrata of mind; that he should strictly and closely study the objective manifestations of mental activity; that he should learn to examine the various limited lesions of the cortex as to area, depth, localised atrophy, relative bulk of convolutions, and tracts of ascending and descending degeneration.

Numerous isolated cases have been reported in which special mental phenomena have accompanied lesions and defects localised in particular regions—cases of lesion of the frontal lobes with affection of the intellect; of other cortical lesions with disturbance of speech and real or apparent mental impairment; of others with hallucinations, visual, aural, tactile, olfactory, and gustatory; of delusion, hallucinatory or otherwise, with arrested or aberrant development of fissures and gyres. In particular, a considerable collection of visual hallucinations and delusions with localised lesions have been reported. Sir J. Critchton-Brown,[1] Spitzka[2] and others, have contributed valuable localisation observations from studies in general paralysis of the insane.

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[1] *Amer. Jour. Med. Sc.*, Phila., 1884, n.s., lxxxvii., 366-391.

[2] *Pacific M. and S. J.*, San Francisco, 1886, xxix., 210-216.

[3] *Brit. Med. Jour.*, London, 1883, ii., 624-628.

Mickle[3] has shown that lesions of the cortical sensory centra of the cerebrum are connected in an intimate way with the production of most of the hallucinations in progressive paresis; that from the cerebral localisation point of view use may be made of the distribution of the cerebro-meningeal adhesions and the cortical changes associated therewith; and that in all cases of visual hallucinations the angular gyre is not affected in the marked manner one would anticipate, on the theory that it is the sole cortical visual centre; nor in cases of auditory hallucinations is the first temporal, viewing it as the sole cortical centre. The morbid anatomy of progressive paresis, he therefore believes, fails to support the exclusive view that these gyres are the sole centres of sight and hearing. The supra-marginal convolution is affected more than the angular in those with visual hallucinations, and the adhesions are often well marked on the posterior parietal lobule. The second temporal gyrus seems to suffer more than the first in cases with auditory hallucinations taken collectively.

Trephining has been performed in many cases of insanity during the last few years, a fair per centage of the operations having been guided at least in part by the principles of localisation.

Two of the recent cases of brain operation, reported by Bennett and Gould,[4] and by Macewen,[5] open a possible new field for surgical interference in insanity. In the case of Bennett and Gould, the patient had received a violent blow on the right side of the head and had a scalp wound without apparent injury to the skull. Pressure on the cicatrix caused the sensation of a flash of light followed by unconsciousness for one or two seconds. The patient had no paralysis, loss of sensation or other symptoms, but was subject to left unilateral convulsions with loss of consciousness, commonly followed by violent mania. The attacks were usually preceded by a bright red flash of light and were succeeded by what appeared to be threatening visual hallucinations. The scar was over the region which corresponded with the angular gyre. A large trephine opening was made by Mr. Gould, bone and dura mater were removed, and exploration was made in different directions in the brain, but nothing abnormal was detected. Five months later the patient was apparently well having had no attack during that time, although for six years before he had had on an average one fit a week. After his recovery he seemed to forget all about the hallucinations. Dr. Bennett, in another case, observed similar hallucinatory phenomena, and after death the angular gyre was found to have been injured. Such cases are of importance, as opening the question of the propriety of excising cortical areas as a method of treatment in insanity as well as epilepsy, when certain subjective phenomena such as hallucinations of sight and hearing can be given a local habitation in the brain.

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[1] 'West Riding Reports,' vol. vi., p. 170.

[2] 'Insanity: Its Classification, Diagnosis and Treatment.' Article on "Paretic Dementia."

[3] *Jour. Men. Sc.*, Oct., 1881; Jan. and April, 1882.

[4] *Brit. Med. Jour.*, Jan. 1st, 1887. [5] *Lancet*, Aug. 11th, 1888.

Macewen's case was one of psychical blindness. The patient had received an injury about a year previously and suffered from deep melancholy and strong homicidal impulses directed against his family, and relieved by paroxysms of pain in the head, of indefinite seat. Prior to receiving this injury he was perfectly free from such impulses and had led a happy life with his family. Behind the angular process was a slight depression which could not account for his symptoms. No motor phenomena were present but on minute inquiry it was discovered that immediately after the accident and for about two weeks subsequently he had suffered from psychical blindness. The angular gyre was exposed for operation, and it was found that a portion of the internal table of the skull had been detached from the outer and had exercised pressure on the posterior portion of the supra-marginal convolution, while a corner of it had penetrated and lay imbedded in the brain. The bone was removed from the brain and re-implanted in proper position, after which he became greatly relieved in his mental state though still excitable. He made no further allusions to his homicidal tendencies.

Cases of double personality and double consciousness, and of unilateral hallucination like the following, reported by Magnan,[1] may eventually receive their proper interpretation through investigations in localisation. Magnan holds that there are hallucinated individuals who hear on one side agreeable things, and on the other side unpleasant. He had had under observation four cases of this kind of which one was reported in detail. The case was one of primary monomania, complicated with epilepsy. On the right side disagreeable statements were made; on the left ambitious ideas were conveyed. These latter hallucinations were obviously secondary to the first. He concluded, first, that these unilateral hallucinations on opposite sides were independent of local lesion; that they did not differ from other hallucinations; that they proved the double action and functional independence of the two hemispheres; that analogous phenomena were noticed in hypnotic states; and that their existence demonstrated the action of separate sensorial centres in the cortex.

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[1] *Jour. de Médicine de Bordeaux*, Sept. 30th, 1883.

Contributions of Cerebral Localisation to General Medicine and Therapeutics.

The vast improvements in precision both in examining and describing the symptoms of nervous disease, and in making and recording the results of autopsies, have been largely due to the stimulus to exactness which has been given by the science of cerebral localisation which has at its very foundation topographical precision.

The contributions of cerebral localisation both to general and local symptomatology, if carefully brought together, would furnish material for an elaborate address. A flood of light has been thrown upon the nature of epilepsy, or rather epilepsies. Many old differential symptoms, some of them once regarded as pathognomonic, have been swept away, and better and surer criteria have been substituted in their place. The clinical teacher no longer announces that unconsciousness is the one sure sign of epilepsy, and the preservation of consciousness of hysteria; but the question of consciousness becomes a relative one in the consideration of both diseases. We are slowly getting the data for a really scientific classification of epilepsy into reflex, toxic, cortical, bulbar and spinal. As Mr. Horsley has recently shown, it is no longer necessary to consider hystero-epilepsy, epileptiform seizures, laryngus stridulus and eclampsia as altogether apart from epilepsy.

Not a few symptom-groups or symptoms formerly not understood at all, and some of them regarded as independent diseases, have been given their proper positions; such affections, for instance, as athetosis, tetany and some spastic diseases of children.

Vagueness has given place to clearness with reference to such affections as cerebral softening; and new light has been thrown upon such common and important diseases as tubercular meningitis, particularly as it affects the convexity of the hemispheres.

Now and then a new experiment or observation on cerebral localisation has let in the light upon some obscure symptom or condition known to the physician. That peculiar perversion of sensory localisation known as allochiria, was noticed for instance by Horsley and Schäfer[1] as the result of lesions produced by them in the limbic lobe.

Something has been accomplished with reference to the action of drugs on localised cerebral areas. I might point to the investigations of Albertoni[2] as to the augmentation of the excitability of the cortex by atropine, and the action of bromide of potassium in reducing the same excitability, a conclusion which has since been confirmed by Rosenbach and others, and is in accord with all clinical experience; to the work of Luciani and other Italian observers on chinconidine and pyrotoxine as epileptogenic agents; and to the experiments of Tamburini, Seppilli, Hitzig and Franck upon the effects of anæsthetics and narcotics on critical areas. Franck[3] has thoroughly investigated the effects of curarization on cortical excitability, and some of his results may prove of medico-legal importance in the study of masked or hidden epilepsy. Danillo, Magnan, and Franck have made important observations on absinthine epilepsy.

Experiments and discoveries like those of Eulenberg and Landois,[4] Wood,[5] Ott,[6] Richett,[7] Aronsohn and Sachs,[8] Wood, Reichert and Hare,[9] and Girard,[10] on the existence and phenomena of heat centres in the brain, have been of practical value in throwing light on the mechanism of fever, and on the action of special drugs and different modes of treatment on forms of high temperature. I will refer very briefly to some of the experiments and inferences of these observers, simply to show their practical tendencies.

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[1] 'Phil. Trans. Royal Soc. of London,' vol. clxxix., 1888, B. pp. 1-45.

[2] Cortical Epilepsy. Experimental Researches. Synthetic Review. By Greuseppe Seppilli. M.D., Alienist and Neurologist, Jan., 1885. Translated by Joseph Workman, M.D., from the *Rivista Sperimentale*, 1884.

[3] *Leçons sur les Fonctions Motrices du Cerveau*, par le Dr. Francois-Franck, Paris, 1887.

[4] *Compt. rend. Acad. de Sc. Par.*, 1867, lxxxii., 564-567. [5] *Op. cit.* [6] *Op. cit.*

[7] *Bulletins de la Société de Biologie*, March 29th, 1884.

[8] *Deutsche Medicinische Wochenschrift*, No. 41, 1882, and *Pflüger's Archiv.*

[9] *Therapeutic Gazette*, vol. ii., 3 s., No. 9, September, 1886, p. 577.

[10] *Arch. de Physiol., norm. et path.*, Paris, 1886, 3 s., viii., 281-299.

Wood for instance holds that with the facts of his experiments in mind, the theory of a causation of fever becomes very plain. "It is simply a state in which a depressing poison or a depressing peripheral irritation acts upon the nervous system which regulates the production and dissipation of animal heat; a system composed of diverse parts so accustomed to act in unison continually in health that they become as it were one system, and suffer in disease together. Owing to its depressed, benumbed state the inhibitory centre does not exert its normal influence upon the system, and consequently tissue change goes on at a rate which results in the production of more heat than normal and an abnormal destruction and elimination of the materials of the tissue. At the same time the vaso-motor and other heat dissipation centres are so benumbed that they are not called into action by their normal stimulus (elevation of the general bodily temperature), and do not provide for the throwing off of animal heat until it becomes so excessive as to call into action by its excessive stimulation even their depressed forces. Finally, in some cases of sudden and excessive fever, as in one form of so-called cerebral rheumatism, the enormous and almost instantaneous rise of temperature appears to be due to a complete paralysis of the nervous centres presiding over heat production and dissipation."

Girard,[1] as the result of certain experiments on rabbits, concludes that the cerebral centre of thermo-genesis is in the corpus striatum. Lesion of the median portion produced well-marked increase of heat, and this was not the result of spasm of vaso-constrictor nerves of the skin. Exciting the region electrically caused a notable increase of heat showing that this resulted from excitation and not from paralysis. Similar excitation caused increase of urea, indicating an increase of combustion in the organism, which was accompanied by considerable emaciation. Girard believes this apparatus or centre increases the heat under excitation and notably influences the regulation and production of heat; also that artificial increase of heat is not identical with that of fever. Increased production and at the same time diminished dispersion of heat, from the body are according to his view the two conditions essential to fever.

One of the latest contributions of Ott is on the heat centres of the cortex cerebri and pons varolii. He found that when in his experiments upon rabbits a puncture was made just in front of the ear into the cortex, there ensued a fugitive rise of temperature; and this observation led him to try in cats the effects of removal of areas of the cortex in this and other regions. A point at the juncture of the supra-Sylvian and post-Sylvian fissures was found to have the highest thermic value. Other parts of the brain, with the exception of the cruciate centres, had but small effect upon the temperature. The rise of temperature after injury to the Sylvian centres was from three to four degrees and continued till the death of the animal which was usually about the fifth or sixth days. The calorimetric investigations showed that either immediately or at the end of twenty-four hours, the heat production and heat dissipation were increased; after that they usually fell below normal although the temperature remained elevated, with a weight decreasing daily. He believes that this increase of heat production was not due to secretory changes as pulse and pressure both rose for a short period, and then fell to a certain extent below normal although the temperature was then rising.

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[1] *Gazetta Degli Ospitali*, Aug. 17, 1887.

The mechanism of temperature production according to Ott is: (1) Thermotaxic centres, cruciate and Sylvian of Eulenberg and Landois; (2) Thermotaxic and thermo-genetic centres—the centre about Schiff's crying centre, and the extra striate (Sachs and Aronsohn), and the thalamic centres; (3) Thermogenetic centres—spinal centres.

"It is probable," says Ott, "that after injury to the cortical heat centre, the basal and spinal thermogenetic centres are temporarily permitted to obtain the upper hand, but that shortly the other cortical heat centres bring the thermogenetic centres into subjection and thus reduce the heat production. In the case of lesion of the basal and spinal thermogenetic centres for a short period they primarily overcome the cortical centres, but finally succumb to the domination of the thermotaxic centres of the cortex. In other words, the Sylvian and cruciate centres constantly antagonise the basal and spinal thermogenetic centres. It is also probable that under certain impulses the cortex and basal centres combine together to antagonise the spinal thermogenetic centres. It would seem that an injury to the thermotaxic or thermogenetic apparatus sets up a fever which is primarily accompanied by increased production and dissipation; but they soon fall below normal, whilst the fever continues till the lesion is repaired. This would lead to the belief that in continued fever the generation of a ptomaine is continuously carried on for some time and thus keeps up the fever."

Scarcely anything as yet has been contributed by these investigations to the surgical aspects of the question; but a case reported by Mr. Page[1] has at least some suggestive value, and is the only one to which I will allude. A man, from a fall, had a wound one inch in length in the right parieto-occipital region. He was put to bed and became dull and apathetic; his temperature rose until it had reached 105° F., but otherwise he presented no symptoms that could be determined. Trephining was performed over the posterior part of the temporo-sphenoidal lobe. The patient's high temperature rapidly subsided and he recovered without other symptoms.

Before leaving the consideration of these questions of general symptomatology and theurapeutics it might be well briefly to refer to what has been accomplished in cerebral localisation with reference to some of the organic or involuntary functions. Are there circumscribed localised areas in the cerebrum which are capable of producing certain so-called organic or involuntary effects, or effects which may be classed as somewhere between the purely voluntary and the involuntary? In other words, to put the question in its simplest expression, have we centres—comparable to those which give definite motor reactions—for such functions as those of respiration, heart-action, vascular tone, oculo-pupillary movements, the secretion of sweat, saliva, and bile or the excretion of urine? No one has studied this subject with the thoroughness and originality of François Franck in his great work on the motor functions of the cortex, to which reference has already been made. His conclusions are based chiefly upon the results of irritation of various regions of the brain. He found excitation of the brain in various regions of the cortex efficient to produce organic and partly organic manifestations, but such areas were not circumscribed and invariably the same. Changes in respiration, arrest or increase of the movements of the heart, flushing or paling more or less local or general, suppression or increasing the flow of saliva, sweat, bile, urine, &c., could all be brought about by experimenting upon the cerebral cortex of dogs and monkeys. Such results however he does not believe should be regarded as simple reactions comparable to the definite movements caused in face or limb by irritation of the centres assigned to these parts. They are complex results more comparable to the reflex effects produced from irritation of sensory surfaces anywhere. He shows that the suppression of the cerebral region, whose excitation so clearly produces organic effects, does not cause the loss of the function put into action by the excitation.

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[1] *Lancet*, London, 1887, ii., 1216.

These views are probably correct in the main, although they may receive some modification with increase of knowledge upon this subject. Regions of the brain in the process of evolution have been differentiated into definitely localised centres of representation in proportion as the functions represented have become more and more volitional, more and more under the control of the individual. We can never probably have localisations for organic manifestations which will be available, for instance, for the purposes of the surgeon. A closer and fuller study may show the truth to be largely the same even for the so-called thermic or heat centres. It is altogether doubtful whether we have distinct vaso-motor cortical centres comparable to the simple centres for motion. In reference to this question Franck says that all localisations of this kind ought to be renounced. The cortical surface, he says, agrees in a certain degree with the sensory surface and does not contain the vaso-motor centres any more than the organic centres, whatever they may be; the cortex fills the roll of separation, and not that of a productive organ of visceral reactions The true vaso-motor centres are contained in the bulb and spinal cord. They receive the cerebral excitations as they receive the peripheral excitations, and react in both cases in a reflex manner in consequence of a similar mechanism.

Such localisations as those of Christiani[1] of higher respiratory centres must not be regarded in the same light as motor, visual and other independent simple localisations. This investigator, as the result of a series of experiments on rabbits and dogs, believed that he had found higher respiratory centres, three in the basal ganglia; first an inspiratory one, chiefly reflex, at the bottom of the third ventricle; second, one, also inspiratory, at a point between the anterior and posterior corpora quadrigemina; third, an inspiratory and inhibitory centre at the entrance to the aqueduct of Sylvius. He also discovered, anterior to the inspiratory centre in the third ventricle, a coordination centre.

To speak of emotional centres in the same sense that we do of motor, visual, or auditory centres, is also nnphilosophical. In certain organic brain lesions says Pontoppidan,[2] emotional manifestations such as laughing or crying appear without a cause; or an emotional cause produces undue effects—a pain, for instance, produces laughter. Such symptoms are usually met with in disease of the pons and oblongata. The investigations of Pontoppidan seem to show that the centres affected in such cases are those in the vicinity of the vaso-motor centre in the pons. He describes in detail three such cases. In the first, any question caused the patient to laugh; in the second, laughing or crying occurred indiscriminately when any attempt at conversation was made by the patient; in the third, fits of laughter occurred without any apparent cause—the mere entrance of any one into the room would produce one. In two of these cases autopsies showed the existence of apoplectic clots in the crus cerebri and pons Varolii, and other symptoms of pons disease were present.

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[1] *Du Bois. Arch.*, 36, 1884. [2] *Contr. f. Nervenheilk*, 1887.

In the nervous wards of the Philadelphia Hospital are several cases similar to those described by Pontoppidan, other symptoms pointing also to disease of the pons. Such facts however do not indicate the existence of a special centre for emotion, comparable in any true sense to the circumscribed centres of the cerebral cortex; but rather point to the fact that in the pons oblongata we have crossing and interblending the various tracts, ascending, descending and transverse, which unite the higher regions of the nervous systems to those lower centres which energise the nerves and muscles concerned in the expression of emotion, or join together these lower centres and the cerebellar hemispheres.

Cerebral Localisation in its Relations to Surgery.

Let me now turn to the surgical aspect of this great subject—the surgical aspect in so far as it concerns the neurologist; it is upon this that the attention of the medical world is riveted to-day.

In this portion of my remarks, I will consider (1) the forms of disease and injury in which cerebral localisation is a valuable aid to diagnosis; (2) the parts of the brain accessible to surgical interference, and the topographical diagnosis for these accessible areas, with some sources of error in diagnosis.

The neurologist is now constantly called upon with the surgeon for the relief of intra-cranial affections long held not to be amenable to treatment, and scarcely worthy, from a practical point of view, of diagnosis. My remarks must be chiefly concerned with questions of diagnosis.

Examination of medical literature shows that operations upon the brain, guided by localisation, have been for tumour, cyst, fracture, abscess, hæmorrhage, and discharging cortical areas.

Brain Tumours.

It may be broadly affirmed that brain tumours should be removed by operation when their exact position can be diagnosticated, when they are in accessible areas, when they are solitary, and when they are not of enormous size. Of Dr. Park's 63 cases, 11 are cases of tumour and 12 of cyst; of 17 operations by American surgeons, 5 have been for tumour. In this connection I will only stop to give some facts and draw some inferences from personal experience; it is often wise to review personal experience even if in so doing we sometimes awaken vain regrets. I have notes of 20 cases of brain tumour with autopsies, most of which have already been published in some form. Hale White's[1] cases numbered 100; and Seguin and Weir,[2] combining the statistics of White and Bernhardt, tabulated 580 cases. Twenty cases are comparatively few, but such a list has the advantage of thorough personal knowledge. Of these 20 cases the locations were as follows: Prefrontal lobe, 2 cases; posterior portion of second frontal gyre, 1 case; motor (Rolandic) zone, 6 cases; superior parietal lobule, 1 case; temporal lobe, 2 cases; cerebellum, 2 cases; mid-base and corpus callosum, 1 case; pons-oblongata, 4 cases; optic thalamus, 1 case.

Twelve out of the 20 cases were in areas accessible to operation; one of the accessible cases was multiple. Of the 11 accessible cases left, 4 were fibromata, 3 gummata, 2 tubercular, 1 a carcinoma, 1 a glioma with intercurrent hæmorrhage. In neither of the 2 tubercular cases would operation have been successful because of the diffusion of cerebral tubercular disease. The carcinoma and glioma would probably have given only temporary success. Of the 7 cases left, all could probably have been removed successfully by operation at some stage of their growth; although in 3 of the cases, at the time of death, the tumours were of such size, and the break-down of brain-tissue in their neighbourhood was so great, that the operation then would probably not have resulted in success. In at least 4 of the 20 cases, operation at any time before death would in all probability have been wholly successful. Is it any wonder that vain regrets for lost opportunities sometimes arise?

I favour the removal of old gummata, and this opinion is based upon considerable experience. Again and again I have seen such growths resist the most active and persistent anti-syphilitic treatment. It is probable that one reason why they will sometimes not yield to medicinal means is because in the progress of their growth they have obliterated blood vessels and become practically inert foreign bodies. Bergmann and White oppose, and Seguin favours the removal of gummata.

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[1] Guy's Hosp., 1884-85, 3 S., xxvii.

[2] *Am. Jour. Med. Sci.*, July, August and September, 1888.

Cranial Fractures.

Localisation rules are sometimes of value, even in cases of visible and easily detectable fractures, with lacerations, scars, clefts, depressions or ridges. These rules may be called in to clear up obscure points. Often in cranial fractures the extent of unseen damage cannot be told by the position and character of visible lesions. Numerous cases have been reported in which the operators would have been misled by trusting to external evidences alone, but in which by calling in the established facts of localisation to assist they were able to place the trephine over the best spot for operation. Examination of surgical literature also shows that in many cases, demonstrated by autopsies, if the rules of localisation had been properly applied, the site of hidden fractures either of the internal table or not, could have been determined and operations performed to the great benefit of the patient.

The best point for trephining, in cases of fracture is not always the place of the greatest depression or cleavage, or over the centre of a large scar. In fracture cases the symptoms of dural irritation will often be prominent, and, particularly when the injury is over the motor area, may confuse the picture of spasm which is presented. The spasm may be dural or reflex rather than cortical, or may have a mixture of reflex and cortical characteristics; and hence may be on the same side as the lesion, or general, and thus involve the mind of the diagnostician in some doubt. An abscess resulting primarily or secondarily from a fracture may be so situated or may have so enlarged that localisation rules alone can determine the best site for trephining. According to Jacobson,[1] out of 70 cases of middle meningeal hæmorrhage a fracture was present in 62, so that in the majority of instances both fracture and hæmorrhage must be taken into account.

Intra-cranial Abscess.

The question of intra-cranial abscess as well as fracture will be fully treated by Dr. Park and I will therefore say but little

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[1] Guy's Hosp. Rep., 1884-85, 3 S., xxviii., 147-308.

Regularly recurring rhythmical movements of the left side of the body. Sensation and consciousness were normal. The right side of the body could be moved at will. Articulation impossible. Incontinence of urine and fæces. The movements of the body continued during sleep, and gradually lessened, leaving the leg on the fourth day and arm on the fifth. A clot as big as a hen's egg was present on the right side of the brain: this was superficial, reaching from the pre-central gyrus to the occipito-parietal fissure, and from the longitudinal fissure to the temporo-sphenoidal lobe.

C[illegible] [illegible]emorrhag
[illegible]ortical H[illegible] [illegible]e.

Sub-dural or in[illegible] hæmo[illegible] e frequently also
cortical, that is, ter-meningeal [illegible]rrhages ar a-arachnoid and
cortex. Occasions hey invade or involve the P nial hæmorrhage
occur which may b lly however cases of intra-cra s cortical or sub-
pial. These are us e more particularly classed a n take place from
arterioles or capill hally limited in size and ofte orsley[1] illustrates
what is meant by aries. A case reported by H l hæmorrhage.

This was the c one of the forms of true cortica ering from tuber-
cular disease o ase of a man who had been suff and suddenly
developed symp the bone for some months, inal sinus with
cortical epile[illegible] ns of thrombosis of the longit[illegible] [illegible]ing, not only
as one of a [illegible] result. The case is in [illegible] because
[illegible] of cerebr[illegible] hæmorrhag[illegible] [illegible] the

half of the middle third of the superior frontal convolution for half its breadth. This, the only lesion in the left hemisphere, was situated at the highest point of the area for the head and neck in the left hemisphere."

Small, superficial, cortical extravasations of this kind are to be localised by the rules and principles for irritative and destructive lesions of the brain surface of whatever character.

Intra-cerebral Hæmorrhage.

Intra-cerebral hæmorrhage will next engage our attention. Of course a hæmo ge may nywhere within the cerebrum—in th orrha ntal, ake place , parietal, occipital or temporal lobe e pre-fro nno postero-frontal fferentiate between the varieties of but we ca stop here to di se positions. The remarks upon th æmorrhages occurring in th any kind in these locations will in e localisation of lesions of hage. In this connection the discus large part apply to hæmorr to those varieties of intra-cerebral hæ sion will be largely confined mmon, and which might be said morrhage which are most co cal—the cases of hæmorrhage int o have become almost classi sses.

Commonly i or near the great ganglionic ma as Gendrin and Charcot ha tra-cerebral hæmorrhage occu the caudate or lentic nted out, not in the bod external phine successfully for intra-encephalic hæmorrhage. This must be done, if at all, at a point where it has been determined by pathological observation that the hæmorrhage in its enlarging waves outwards usually comes nearest the surface, or would be most easily reached and relieved. The cases of hæmorrhage in which the ventricles are broken into and inundated would probably be benefited only very rarely by operation, but no harm could be done in such an almost necessarily fatal case.

Intra-cerebral hæmorrhage may occur in any one of half a dozen positions with reference to the three great ganglia at the base of the brain, and the internal or external capsule. With our present knowledge [illegible]tion of some of these hæmorrhages cannot from [illegible] ta[illegible]ly determined. It remains true n[illegible]s ago, that lesions confined t[illegible]en the plete according to the extent of the hæmorrhage; stertorous respiration, sometimes so far as the mouth is concerned, one-sided; sometimes also Cheyne-Stokes; temperature at first lowered and afterwards rising; pulse sometimes slow and full, sometimes weak and intermittent. Conjugate deviation of the head and eyes may be present but is not invariable; it is usually away from the side of the paralysis. It is not infrequently somewhat difficult to determine the full extent and character of the paralysis and loss of sensation, if this also be present, in these cases of apoplexy. Careful inspection of the face however will usually show some drooping on the side of the paralysis and some pulling to the other side. Watching the lim [illegible] lysed members will be seen to be [illegible] pa [illegible] ly. The paralysed extremities [illegible] limp and offer no resistan [illegible] is offered by the

the right, diminished on the left. His head temperature, taken once at a spot corresponding to a point just below the middle of the horizontal branch of the Sylvian fissure, was 96·2° on the left, and 100·4 on the right. The patient lived six days from the time of his admission. His body temperature when first taken was 96°. It rose the second day to 101°, and from that time on until his death, ranged between 99° and 102°, being at the highest point at the time of death. He developed pneumonic symptoms three days after admission.

At the autopsy, on exposing the left lateral ventricle, a nearly black, irregularly shaped spot was seen reaching across the caudate nucleus where it begins to curve around the thalamus. This appearance indicated a recent clot which had not quite broken into the ventricle, still having a thin roof formed by a layer of the caudate body. The ganglia and capsules were studied by transverse sections. The anterior limit of the extravasation was towards the median line of the brain, and was three-fourths of an inch from the head of the ganglia. Its posterior limit, a narrow wedge, was one-third of an inch in front of the posterior extremity of the thalamus. The blood was still fluid and the parts involved by the clot were chiefly the middle portions of the lenticular body and internal capsule, and an external anterior segment of the thal- ia mater of the convexity was œdematous and c anc yperæmic, and Pacchyonian gra blood vessels were highly at d interstitial nephritis. On atch of con-more harm than good might be done to the patient. The peculiar position in which the ganglia and capsules are located with reference to the Sylvian fossa, the island and the descending horn of the ventricles, would constitute one of the chief sources of difficulty in attempting to trephine for intra-cerebral hæmorrhage. Still the operation is not impossible, and we will probably eventually learn exactly how far it can be resorted to with advantage, probably only in a very limited number of well chosen cases.

Intra-cerebral Hæmorrhage with Inundation of the Ventricles.

What now are the symptoms of intra-encephalic hæmorrhage with ventricular inundation? Whether this form of hæmorrhage is or is not susceptible of improvement by operative interference, its diagnosis has considerable negative practical importance. I have, for instance, known the diagnostic question chiefly discussed in an important case to have been, whether the patient was suffering from hæmorrhage which had burst into the ventricles, or from supra-dural or sub-dural clot of immense size. Certainly as I have seen the cases there oints of resemblance
between some vent me of meningeal
hæmorrhage; are sufficient to
separate the v l and minute in

right than of the left limbs; he occasionally moved the left arm and leg, and a tendency to contracture was present on the right side. Tremulous and spasmodic movements occurred on both sides of the body, but were a little more marked on the right than on the left. The pulse on the left side was comparatively full and strong; on the left feeble frequent and irregular. The temperature was taken several times in both axillæ, and varied between 99° and 101·2°, but with no uniformity as to the two sides. A marked difference between the head temperature of the two sides was noted, the right Rolandic station giving temperature of 102, the left only 99·2. The breathing passed through three periods, at first it was puffing, soon Cheyne-Stokes, and two hours before death regular but constantly feebler and shallower. When of the Cheyne-Stokes type, the period of nearly regular breathing lasted from four to five minutes, the apnœal stage only from eight to fifteen seconds. When breathing began after the apnœa it presented an ascending character, but the apnœal stage began very abruptly. He died about twelve hours after the stroke, and before death the paralysis of the limbs and face became absolutely general. The pupils became more dilated but not unequal.

Autopsy.—Resting the brain on its convex surface, large masses of dark blood could be seen occupying the central region of the base from the pons to the optic chiasm; the blood enveloped the cranial nerves in this area, and infiltrated the membranes and the spaces beneath them far out into the Sylvian fissure. Hæmorrhagic foci were found here and there in the pia of the cerebellar hemispheres the substance of which showed a few bloody points. The fourth ventricle was filled and distended with dark blood; its floor showed a very slight depression or splitting at the upper part; the aqueduct of Sylvius was very greatly dilated. The lateral ventricles which were entered from below, were filled with blood; their cornua were also enormously distended with blood. The septum lucidum, fornix, corpus callosum and commissures were broken down, and the lateral and third ventricles had become one cavity engorged with blood. The anterior extremity of the left optic thalamus and the cue-portion of the caudate nucleus were broken through. The hæmorrhage had apparently taken place either from one of the lenticulo-optic or one of the posterior internal optic arteries.

Certain points of difference are to be noted between this case and the previous one, in which the hæmorrhage did not reach the ventricles, as for instance the more sudden and profound unconsciousness, the complete unilateral paralysis which soon became general, the absence of all sensory response, the tremulous and spasmodic movements of both sides of the body and the peculiar Cheyne-Stokes breathing.

I have examined the specimen from one case of secondary ventricular hæmorrhage in which the primary extravasation took place in the centrum ovale of the parietal lobe, the blood breaking through the roof of the ventricle; but usually the secondary ventricular flooding takes place in the manner and from the direction indicated in he account of the case just given.

Of primary ventricular hæmorrhage I have had no experience. "Primary ventricular hæmorrhage," Gower says, "causes symptoms which may from the first closely resemble those of the secondary form, but more frequently the onset resembles that of hæmorrhage into the substance of the brain, in the presence at first of unilateral symptoms. Prodomata are rare, but headache is occasionally met with, very variable in seat, character and duration. The onset may be (1) By sudden apoplexy, deepening rapidly; death may occur in a few hours. (2) By apoplexy with hemiplegic symptoms, or with convulsions. (3) In the very rare slow hæmorrhage, hemiplegia first occurs alone, loss of consciousness only supervening after a few hours. Hemiplegia occurs because the blood is effused into one lateral ventricle and causes paralysis on the opposite side by the compression of the motor path or centres. When the effusion is rapid and both lateral ventricles quickly become distended, the unilateral symptoms quickly give place to general relaxation of the muscles and loss of all reflex action. Rigidity is often met with, but less frequently than in the secondary form; it is usually bilateral, sometimes one-sided, and occasionally involves only the muscles of mastication; it is often intermittent. Convulsions are also frequent, occurring in at least a third of the cases, sometimes general, sometimes affecting only the paralysed side, or only part of it. In cases of slow onset, speech is often lost before consciousness. The power of swallowing usually persists until the apoplexy becomes profound. The temperature resembles that of other forms of cerebral hæmorrhage. The malady is usually fatal, but recovery has occurred, as is proved by old and altered clot being sometimes found in the lateral ventricles, but it is possible only when the hæmorrhage is small in quantity and the symptoms are slight and equivocal." The fact that recovery has occurred in such a case is a reason for considering the practicability of trephining.

Tapping and draining the ventricles have been performed, though rarely; but in the future, with the comparative immunity from danger in our present methods of attacking the brain, may be resorted to much more frequently. The ventricles can be reached with precision at several points, best probably from an anatomical and surgical point of view, by way of the posterior horn, or perhaps where the lateral ventricle and the middle and posterior horns diverge. Besides blood, effusions into the ventricles may be also either serum or increased cerebro-spinal fluid, or pus from an abscess.[1]

Various practical questions arise in connection with the subject of trephining for intra-cerebral clots, particularly when deeply situated. It has been suggested that it might be impossible to remove the extravasation on account of its having formed a firm coagulum. It does not always do this. Within one week I saw two cases of intra-encephalic hæmorrhage, in one of which the cavity was filled with a firm clot, and in the other the blood was entirely fluid, although the patient had been dead more than twenty-four hours. Why this difference should occur I do not know, but it is a fact well known to surgeons that in hæmatocele, no matter where situated, when not in contact with the air, the blood is sometimes coagulated, and sometimes is not. Even though the blood has coagulated it might in some cases be removed by carefully enlarging the opening made by the knife to reach the seat of hæmorrhage with flat retractors, and then extracting the coagulum in fragments with forceps or a spoon. The bleeding in case of cerebral hæmorrhage is probably stopped because of the retraction of the vessel and the forming of a small coagulum in it, but of course the danger of producing a fresh or renewing an old hæmorrhage should be considered. If such operations are resorted to, care should be taken not to move the patient more than is absolutely necessary.

Cortical Epilepsy without Gross Lesion.

In cases of cortical epilepsy when the symptoms indicate a discharging lesion of a localised cortical area, operation is justifiable whether or not the probability of a gross lesion can be made out. Hughlings Jackson in the course of a discussion of a paper on brain surgery, read by Mr. Horsley[1] at the meeting of the British Medical Association at Brighton in 1886, strongly advocated the cutting out of the part of the cortex which represented the peripheral parts first in the spasm, whenever the spasm began very locally and deliberately, and when the fits were often repeated. He advocated this, no matter in what condition the brain cortex might be found. He considered it quite certain that epileptiform seizures would be impossible in such a case if enough of the so-called motor area were removed. He believed it better to have some permanent paralysis than to be subject to fits, some becoming universal. This advice has already been acted upon by Horsley,[2] Keen,[3] Lloyd and Deaver,[4] and Hearn and the writer. The most interesting case of the kind yet reported is that of Lloyd and Deaver. Macewen rather advises against this operation, particularly if large wedges of brain tissue are to be taken out, but I believe it to be good practice, even some permanent paresis being preferable to epileptic attacks with their destructive effects on the brain.

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[1] Since the meeting of the Congress, Dr. W. W. Keen, of Philadelphia has proposed tapping and draining the ventricles as a definite surgical procedure, describing an operation for this purpose. He says: "As we now open the belly and drain in tubercular peritonitis with such remarkable success, I would propose that we do precisely the same for the brain. That it may be done with precision and without serious injury to the cerebral tissues the history of the present case, I think, abundantly shows; that it is even *more* urgently necessary in the brain than in the chest or belly seems clear when we consider the relative effects of pressure in the two cases. In the chest or belly the walls are more or less yielding or spongy, to a large extent. They can bear great and long continued pressure but with little damage to their ultimate integrity or to life, if the pressure be relieved within any reasonable time.

Not so in the cranium. The walls are rigid bone, and the brain can undergo but little pressure, and for a brief time (except it be gradual, as in chronic hydrocephalus) without inviting death. The fatal issue is so uniform that *any* means that holds out a reasonable hope of relief, even though it involves great risk to life, should at least be tried; and the proposal in the present paper seems at least to involve but a moderate danger to life with a reasonable probability of success." (*Medical News*, December 1, 1888.)

Accessible Areas of the Brain.

More and more has that region been narrowed which cannot be reached by the venturesome surgical explorer. The lateral aspect of the pre-frontal lobe, the entire motor area, the superior and inferior parietal lobules and the upper temporal region can of course be attacked with the greatest facility. In the regions difficult yet possible of access, lesions of large size and of displacing character will be more readily reached. The orbital surfaces of the pre-frontal lobe can be reached and large displacing lesions removed by trephining low down in the frontal bone. In Durante's case the tumour removed occupied the left anterior fossa of the cranium. Almost the entire temporal lobe with the exception of the parts bordering on the mid-brain, is accessible. The occipital lobes have been operated upon successfully. With care the great median fissure may be entered for lesions of the marginal convolutions and limbic lobe. The longitudinal sinus has been successfully plugged and ligated. The outskirts of the ganglia have been approached, and the ventricles have been pierced. Even a tumour situated on the intra-cranial portion of the auditor and facial nerves can probably be reached and removed. Suckling and Jordan,[1] Bennett May,[2] Horsley,[3] and Weir have looked during operation with the eyes of the flesh on the foramen magnum itself. Absolutely inviolable then are only the middle region of the base, and its bordering convolutions, the corpora quadrigemina, and pons-oblongata.

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[1] *Brit. Med. Jour.*, Lond., 1886, ii., 670-675.
[2] *Ibid.*, Lond., 1887, i., 863-865.
[3] *Am. Jour. Med. Sci.*, vol. xcvi., No. 4, Oct., 1888.
[4] *Ibid.*, vol. xcvi., No. 5, Nov., 1888.

In the accessible areas of the brain are (1) regions in which an absolute localisation can be made by positive symptoms; and (2) regions in which a close approximate localisation can be made by positive symptoms, combined with methods of exclusion and differentiation. Under the first head, come the motor, visual, and motor speech areas and tracts; under the second, the cerebellum, the pre-frontal, and the temporal lobes, with their more or less positively determined functions. The areas for general sensation are still doubtful, but will be considered.

Motor Localisation. Researches of Ferrier and of Horsley and Schäfer.

Motor localisation has become almost an exact science. Properly interpreted, the phenomena produced by irritative and destructive lesions of the cortical motor area can be relied upon to lead the neurologist to a precise topographical diagnosis with as much certainty as the stethoscope for cardiac diseases guides the thoracic diagnostician.

The latest physiological researches bearing upon this are those of Horsley and Schäfer.[4] They give a new diagrammatic representation of the subdivisions of the motor area in the monkey, both upon the lateral and median aspects of the hemisphere, to which I call attention (Fig. 1 and 2). Excitation of the external surface of the hemisphere, in the hands of these experimenters yielded results which were generally similar to those described by Ferrier, which they extended and confirmed, but with some extension as to detail. Comparison of these diagrams with the earlier diagrams of Ferrier, will show the direction in which recent experimentation has added to our precision in motor localisation. (Fig. 3, 4 and 5.)

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[1] *Lancet*, October 1, 1887.

[2] *Lancet*, April 16, 1887, vol., i., p. 768.

[3] *Brit. Med. Journ.* 1887, vol. i., 865.

[4] *Op. Cit.*

In glancing at these more recent results in motor localisation, I cannot refrain from paying a passing tribute to the enduring value of the researches of Dr. Ferrier. Their accuracy and reliability are shown by the fact that the results obtained, even as to detail, have been in the main confirmed by the most careful later investigators. With reference to certain questions in dispute, as for instance, the situation of the area of representation of movement of the head and eyes in the second frontal convolution and adjoining regions, the existence of distinct centres or areas for the senses of touch, pain, and temperature; and the relation of the so-called angular gyrus to vision, his positions have not been seriously disturbed; at the most, it has only been necessary to modify and enlarge his views, as, for example, to admit the part taken by the gyrus fornicatus in sensation, and of the occipital lobe in vision.

On the diagrams of Horsley and Schäfer are placed the names of the zones and centres as determined by them. Below are given the explanations of the circles numbered on the diagrams of Ferrier representing both the monkey and the human brain. The numbering of the centres or areas is the same for both. The diagrams and descriptions are taken from Ferrier's treatise on the 'Functions of the Brain.'

(1) placed on the posterior central and postero-parietal lobule, indicates the position of the centres for movements of the opposite leg and foot, such as are concerned in locomotion.

(2), (3), (4) placed together on the convolutions bounding the upper extremity of the fissures of Rolando, include centres for various complex movements of the legs and arms, such as are concerned in climbing, swimming, &c.

(5) situated at the posterior extremity of the superior frontal convolution, at its junction with the ascending frontal, is the centre for the extension forwards of the arm and hand, as in putting forth the hand to touch something in front.

(6) situated on the ascending frontal, just behind the upper end of the posterior extremity of the middle frontal convolution, is the centre for the movements of the hand and forearm, in which the biceps is particularly engaged, viz.: supination of the hand and flexion of the forearm.

(7) and (8) centres for the elevators and depressors of the angle of the mouth respectively.

(9) and (10) included together in one mark the centre for the movements of the lips and tongue, as in articulation. This is the region, Ferrier says, disease of which, on the left side, causes aphasia, and is generally known as Broca's convolution. (It will be seen later that I regard these as oro-lingual centres, but place another propositionising speech centre in advance of this area.)

(11) the centre of the platysma, retraction of the angle of the mouth.

(12) a centre for lateral movements of the head and eyes, with elevation of the eyelids and dilatation of the pupil.

(*a*), (*b*), (*c*), (*d*) placed on the ascending parietal convolution, indicate the centres of movements of the fingers and wrists.

Circles (13) and (13) placed on the supra-marginal lobule and angular gyrus, indicate the centre of vision, which includes also the occipital lobe.

Circles (14) placed on the superior temporo-sphenoidal convolution, indicate the situation of the centre of hearing.

The centre of smell is situated in the uncus gyri hippocampi or hippocampal lobule (Fig. 5, V).

In close proximity, but not exactly defined as to limits, is the centre of taste.

The centre of touch is situated in the hippocampal region (Fig. 5, H) and gyrus fornicatus (Fig. 5, Gf).

Physiological Experiments on the Human Brain.

New clinico-pathological facts obtained from surgical operations, and justifiable physiological experiments made upon the brain during such operations, have all helped to more accurately fix the sub-areas of the motor zone. In a number of operations on the motor cortex weak faradic currents have been used to accurately localise and define the centres sought. In four instances I have seen experiments of this kind and in one had excision of the cortex performed through the indications thus offered. I have also had the opportunity of observing the effects of faradising the white matter beneath the excised human cortex. Brief reports of such experiments occur in accounts of operations by Horsley, Keen, Weir and Seguin, Lloyd and Deaver, and others. Horsley first resorted to this means of diagnosis nearly five years ago. The neurologists are thus to some extent repaying in kind the gifts received from physiology. Such experimentation is not only justifiable, but sometimes demanded in the interest of the patient. Gentle faradisation of the human cortex does no harm, although it is not so certain that this is true of the application of the galvanic current. The light thrown upon disputed questions by close repeated examinations made after operations will be referred to later.

Boundaries of the Motor Areas.

Let us now glance at the boundaries of the various motor areas—in front, behind, above, below. The anterior branch of the Sylvian fissure extended mentally may be regarded as defining the anterior limit of the motor area, including the centres for emissive speech and for the head and eyes. The area which represents the movements of the face is somewhat accurately limited in front by the precentral fissure; but the movements of the upper extremity have their representation more forward of this line, as do also those of the lower extremity. In front, indeed, the region for the representation of the upper limb extends into the mid-frontal gyre for perhaps one-fourth of its antero-posterior extent; blending in the anterior portion of this forward extension with the region for the head and eyes.

The inter-parietal (intra-parietal) fissure is usually regarded as forming the posterior limit of the motor area. This large fissure runs upward and backward across the parietal lobe: It is doubtful whether in man the whole of the superior parietal lobule or convolution is concerned with motion, and hence the so-called retro-central fissure is perhaps the more probable posterior boundary of the true motor region, the postero-parietal area being concerned, in part at least, with sensation.

This so-called retro-central fissure (Fig. 6, Rc) is practically very constant in the human brain and has been regarded by Wilder and others as a distinct sulcus. I have in a few instances seen it of nearly the same length and depth as the central fissure itself. It is regarded by some as a secondary upward extension of the anterior extremity of the inter-parietal fissure. It generally runs parallel with the upper two-thirds of the central fissure, very clearly bounding behind the posterior central gyre. For practical purposes of operation, at least, this retro-central fissure may be regarded as the posterior boundary of the motor area, rather than the inter-parietal fissure as commonly described. This would leave a distinct postero-parietal region on the lateral surface of the brain in *man*, of uncertain function—a region included between the retro-central fissure in front and parieto-occipital behind.

The horizontal *branch* of the Sylvian fissure forms, as is well-known, the inferior boundary of the motor region.

Until quite recently the longitudinal fissure or median edge of the hemisphere was generally regarded as the superior boundary of the motor area, but the investigations of Horsley and Schafer have shown that this area extends over the edge of the hemisphere into the so-called marginal convolutions on the mesial aspect of the hemisphere, as represented in the diagram (Fig. 2).

As these results are not generally known, it might be well to quote from these authors their general conclusions as to motor representations in the marginal gyres.

"Looking, as a whole, at the results of stimulation of the excitable portion of the marginal gyrus," they say, "it would appear that the application of the electrodes at successive points from before backwards produces (1) movements of the head; (2) of the forearm and hand; (3) of the arm at the shoulder; (4) of the upper (dorsal) part of the trunk; (5) of the lower (pelvic) part of the trunk; (6) of the leg at the hip; (7) of the lower leg at the knee; (8) of the foot and toes." These movements, they say further, in a foot-note, are the primary movements, but as will be seen from previous descriptions, they are almost invariably complicated by secondary movements, which are usually the primary movements produced by excitation of the adjacent parts. The part of the marginal convolution which is concerned with the movements of the leg and foot is that portion which is often known as the para-central lobule.

Diagrams of the Areas and Sub-Areas of the Human Brain.

Based upon the investigations of Ferrier, Horsley and Schäfer, and others, and upon a study of cases, personal and collected from the literature of the subject, the diagrams (Fig. 6 and 7) have been made to approximately represent the areas and sub-areas or centres in the motor zone. In addition, as far as possible, I have indicated areas or centres for other functions—speech, vision, hearing, &c.—so as not to necessitate the repetition of diagrams.

These diagrams (Fig. 6 and 7) approximately indicate the views held by most localisationists, as the result of experiment and its confirmation or modification by clinico-pathological observation. They represent the division of the lateral and median surfaces of the cerebrum into higher psychical, motor, sensorial, visual, auditory, olfactory and gustatory areas; also the sub-division of the motor area into sub-areas, for speech, the head and eyes, the face, arm, leg and trunk; and the further sub-division of these sub-areas into centres for certain specialised movements of the face, arm and leg. The diagrams for the motor sub-areas are based upon the diagrams and researches of Horsley and Schafer, but with some modifications as to extent and arrangement. Although a large portion of the paper immediately following is devoted to a consideration of the division and sub-division of the cortex into areas and centres of representation, it will probably serve a good practical purpose to give here immediately in connection with the diagrams a general description and explanation. Only certain main fissures have been indicated by lettering, so as not to confuse: *S*, fissure of Sylvius. *R*, fissure of Rolando or central fissure. *Pc*, pre-centralor vertical frontal fissure. *Rc*, retro-central fissure, sometimes regarded as a secondary branch of the inter-parietal. *F*1, first or superior frontal fissure. *F*2, second or inferior frontal fissure. *Cm*, calloso-marginal fissure. *Ip*, inter-parietal fissure. *Po*, parieto-occipital fissure. *T*1, first temporal or parallel fissure. *Ca*, (Fig. 7) calcarine fissure.

The pre-frontal lobe, that portion of the brain anterior to the universally recognised motor region, has been designated as the higher psychical area. This term is certainly open to objection, but it is difficult to substitute it by any appropriate general expression. All portions of the brain are concerned with processes of mentation, but this pre-frontal region, as Ferrier and others have shown, seems to be related to the highest mental processes, its lesions causing when sufficiently extensive a mental deterioration which is essentially or mainly a defect of the faculty of attention.

The motor area on the external surface of the hemisphere is made to include the posterior portions of the first, second and third frontal, and both ascending or central convolutions, but not to reach backwards so as to take in the superior and inferior parietal convolutions. The sub-divisions of the motor zone into sub-areas and centres are indicated by the wording on the diagram, and are explained more at length in the body of the paper. Following Horsley and Schäfer's conclusion from physiological experiment the areas for the arm and for the head and eyes are made to extend forward and upward to the median edge of the hemisphere, but few if any clinico-pathological observations support this view which is based upon physiological experiment and is probably correct. While therefore the portions of the first frontal convolutions marked with asterisks * * may be regarded as theoretically included in the areas for the arm, and for the head and eyes, we are not justified for operative purposes in extending these areas above the first frontal fissure. No subdivision of the head, arm, trunk and leg areas in the marginal convolutions on the mesial surface of the hemisphere have been made in the diagrams, as these could only be so far as our present knowledge goes, a reproduction of the sub-divisions given by Horsley and Schäfer in their diagram (Fig. 2).

By the sensorial area is meant that for the senses of touch, pain and temperature, and modification of these senses, and it has been made to include the gyrus, fornicatus, hippocampal convolution, precuneus, and also portions of the superior and inferior parietal convolutions. This sensorial area has therefore been extended to the external surface of the cerebrum so as to include the general postero-parietal region. This keeps the motor and sensory areas distinct, and is based upon the reports of cases with autopsies in which marked disturbances of sensation have been present, although experiments on the lower animals do not seem to have differentiated a sensory area in this lateral external region of the brain. The more elaborate development of the human brain in this region must not be lost sight of in considering this question. It is probable that the exact limitation of the area of common sensibility in the cerebrum has not yet been determined; but anatomical and morphological observations as well as clinico-pathological facts, point to the separation of this sensorial area from the motor region by the great callosc-marginal fissure on the median surface, and on the lateral aspect by it and the so-called retro-central fissure, *Rc*, the parieto-occipital fissure sharply demarcating it behind.

The visual area is represented in the two diagrams so as to take in all of the occipital lobe and adjoining portions of both the temporal and parietal lobes including the so-called angular gyre. Such a delimitation brings into fair accord the findings in reported autopsies, and the researches of Ferrier, Munk, Schäfer and others.

Auditory localisation is still in an uncertain state, but limited pathological evidence favours localising this faculty, as Ferrier advocates, in the first temporal convolution, and probably also in the adjoining second temporal.

The views of Ferrier have been accepted as to olfactory and gustatory localisation, according to which the centre for smell is located in the uncinate gyrus, and the sense of taste is closely related to that of smell, and may therefore be provisionally placed in the adjoining fourth temporal convolution. Possibly it is farther back in the temporal lobe than has been indicated in the diagram.

An area including the middle region of the temporal lobe—the third temporal convolution, and adjoining portions of the second and fourth—has been designated provisionally as the ideational centre or region. This is in accordance with the views of Broadbent,[1] Kussmaul,[2] and some others. I believe the ground taken by these authors is a correct one. The only question in my mind is as to the exact localisation of these centres for which they claim a dwelling place somewhere on the sensory, or receptive side of the nervous system. A consideration of the differentiation and localisation of an ideational or conceptional area in the cortex comes up more particularly in discussion of disturbances of speech, and will doubtless be treated of fully by Dr. Starr, whose views may differ from mine. It is necessary, however, briefly at least to discuss the question in attempting a division of the surface of the brain into general areas, and therefore I touch upon it in this connection. This region is according to Kussmaul that portion of the cellular net-work of the cortex in which ideas are produced as a result of impressions of the most varied description made on the senses (object-and-word-images). According to Broadbent also, the formation of an idea of any external object is the combination of the evidence respecting it received through all the senses; and for the employment of this idea in intellectual operations, it must be associated with and symbolised by name. The structural arrangement connected with this process he supposes to consist in the convergence from all the perceptive centres of tracts to a convolutional area which may be called the Idea Centre or Naming Centre. This he believes is on the sensory, afferent or upward side of the nervous system; its correlative motor centre being the propositionising centre, in which names or nouns are set in a frame-work for outward expression, and in which a proposition is realised in consciousness or mentally rehearsed. The destruction of this centre among other things would cause the loss of the memory of names or nouns. As a provisional guess, Broadbent placed this centre in an unnamed lobule situated on the under surface of the temporo-sphenoidal lobe, near its junction with the occipital lobe, as he believed, fibres from all the convolutions in which perceptive centres have been placed by Ferrier, converged to and end in the cortex of this region. It would certainly seem probable that either in this middle temporo-occipital region, or in the insular or retro-insular convolutions, this conceptional, ideational, or naming region is located. Let no one be misled by this use of the terms ideational, conceptional, &c., and charge that it is an attempt to locate the mind in a limited region of the cerebrum. It is only an effort towards a more thorough understanding of the mechanism of thought and speech. A very careful study of the entire subject of speech disturbances, including an analysis of cases already reported, will, I think, be convincing as to the necessity of a higher area for speech and thought, intermediate between the sensory or receptive centres, and the motor or emissive.

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[1] *Brain*, January, 1879.

[2] Ziemssen's 'Cycl. Pract. Med.,' Am. ed., vol. xiv.

From a study of these diagrams it will be seen that it might be practically convenient to sub-divide the brain into five lobes, four of these, at least, according to the great general functions subserved, these lobes having in nearly all directions well defined fissural boundaries. (1) A *higher psychical or inhibitory lobe,* in front of the basal and anterior branches of the great Sylvian fissure and on the median surface in front of the anterior bend of the calloso-marginal fissure. (2) A *motor lobe,* including the posterior parts of the first, second, and third frontal, both ascending or central convolutions, and the adjoining marginal gyres on the median surface. (3) A *lobe for general or common sensation,* including the gyrus fornicatus, the hippocampal convolution, the precuneus, and the postero-parietal gyres. (4) A *lobe of the special senses,* including the whole of the occipital and temporal lobes. (5) The island of Reil or insular lobe.

Differing views have been advanced as to the function of the island of Reil. With its adjoining parieto-temporal convolutions, it forms a distinct lobe, and is, as is well known, sometimes called the central lobe, or the lobe of the insula. Its importance and size are possibly often not fully considered by the physician and surgeon, owing to the fact that in the average human brain it is so thoroughly concealed by the overhanging fronto-parietal convolutions and the temporal convolutions overlapping from below. The position, relations, and considerable size of the insula can be best seen in some of the brains in which development is arrested or aberrant, as in those of the negro, some criminals, and in the idiotic. Thus studying the lobe, it can be seen to be a great intermediate or binding lobe, probably connecting the other lobes of the brain, so that their associated and related functions may be properly performed, and also for the same purpose uniting the ganglia with the different lobes.

Case of Trephining for Cortical Epilepsy.

Before entering upon the discussion of the subdivisions of the cortical motor zone, I will give the details of a case in which the principles of localisation were called in to determine the position of operation. One object of introducing the history of the case here is because in fixing the position for excision of the cortex the faradic current was used, and certain results were obtained which assist in indicating the exact site of certain sub-centres of the motor zone, as for instance those for turning the head, for extension and flexion of the fingers and hand, and for drawing upwards and outwards of the angle of the mouth.

M., fourteen years old, when two years of age had a series of convulsions coming and going during twelve hours, and followed by a stupor which lasted several days. Six years later he again had a series of severe spasms, the epileptic status continuing for several hours; he had a third similar attack about one year later. Since, during the past five years, he has had fifteen to twenty spasmodic seizures, the intervals between them having grown shorter, so that recently they had only been a few weeks apart. Before the convulsions he was usually nervous and excitable, and on coming out of them nearly always complained of pain above and somewhat in front of the left ear. He had always been of an excitable temperament; and unusual excitability was noticeable during the six years between his first and second attack of spasm. He was, however, a bright boy, of good disposition, affectionate and careful of himself, and his general health, as a rule, was excellent. He was seen in consultation with Dr. Wilson Buckby.

Twelve days before he went into a severe convulsion, and from that time he had not spoken, and had had violent spasms with intervals, in the course of twenty-four hours having ten or more distinct paroxysms. In the intervals between the attacks, he was sometimes stuporous and sometimes in a condition of excitement, but his mind was continually clouded so that he did not appreciate his surroundings.

In every seizure of the series of spasms the convulsive movement began the same way; the fingers of the right hand first flexed, then flexion took place at the wrist and elbow, and the parts remaining flexed, soon the whole arm, forearm and hand were drawn upward and somewhat outward. As one of his family expressed it, "his right arm was drawn until it looked like a chicken's wing." His face and head, after the movements in the upper extremity were well under way, were drawn a little to the right, his leg at about the same time, as nearly as could be determined, taking part in the spasm, semi-flexing at the knee, and the toes and foot contracting. The signal symptom was always the same, namely, a movement of flexion of the fingers; and the spasm was always first and most marked in the right upper extremity; it was commonly unilateral, but sometimes became general. Between the paroxysms his right arm was often the seat of a tremulous vibratory movement. Examination in the interval between two seizures showed slight paresis of the lower portion of the right side of the face and more marked paresis of the upper extremity, particularly of the forearm and hand. This was always more decided after each convulsive attack. Although right-handed he constantly used the left hand in preference to the right. Tactile sense could not be closely studied but he undoubtedly appreciated sensations of touch, pain, temperature. Knee-jerk was somewhat exaggerated on the right side.

After several consultations it was decided to trephine, and if no gross lesion was discovered, to excise the cortex of the area or centre for the fingers and hands in the left hemisphere, because of the invariability with which the spasmodic symptoms began in the fingers and hand of the right side.

The trephining was performed July 28, 1888, by Dr. W. J. Hearn. At the operation were present, besides the writer and operator, Drs. W. Buckby, R. B. Burns, J. H. Lloyd, A. H. P. Leuf, W. M. Coplin, M. Imogene Bassette, J. C. Cooper, and C. P. Noble. The head was shaved and prepared anti-septically. A sublimate solution was used and great care was taken with the instruments, sponges, &c. The line of the fissure of Rolando was determined by the methods of Hare and Thane, and a point was selected for the centre of the first trephine at what was considered to be the junction of the arm and face area, about three-quarters of an inch in front of the fissure of Rolando. A large horseshoe flap was made, its convexity backwards. Two trephine openings were made and bone cut away until finally the opening measured in its greatest diameter, which was from above downward, 2½ inches, and 1½ inches in its greatest width. The long axis of the opening was nearly in a line with the general direction of the fissure of Rolando. The region intended to be exposed was the lower two-thirds of the arm area, the upper anterior portion of the face area, the hinder upper part of the speech area and a posterior strip of the area for movement of the head and eyes ; the convolutions uncovered were, therefore, presumably nearly the lower halves of the two centrals, the posterior extremity of the second frontal, and the posterior superior corner of the third frontal. No lesion of the bone or of the dura mater was found. On raising the flap of the dura mater, the pia arachnoid in the lower half of the opening was decidedly œdematous. No gross lesion could be found on inspection and close examination in the pia mater, cortex, or sub-cortex.

Careful examinations were made with the faradic current applied to the cortex with the view of locating the proper centres for excision. Four distinct responses in the shape of definite movements were obtained after several trials; these were (1) in the most anterior position at which movements resulted distinct conjugate deviation of the head to the opposite side; (2) a little below and behind this point, drawing of the mouth outwards and upwards; (3) above this spot for movements of the angle of the mouth, about half an inch, extension of the wrist and fingers was produced; (4) behind and above the latter point, distinct flexion of the fingers and wrist. Continuing and increasing the faradic application at this last determined point, the fingers, thumb, wrist and forearm were successively flexed, and the whole extremity assumed the "wing-like" position, the order of events, according to three persons who were present, and who had observed the patient's spasms, being exactly that which had been noticed in the beginning of his convulsive seizures.

As no gross lesion was discovered on careful examination and exploration, excision was performed of the cortex and sub-cortex so as to include the area excitation of which by faradism started the spasmodic movements of the fingers and wrist. The operation was concluded after the usual manner. The patient recovered from the operation without serious symptoms. In three days his mental condition was much improved; the restlessness, irritability, and semi-maniacal condition which had been present before the operation passed away. His aphasia persisted. He had distinct paresis of the fingers and wrist, including the thumb, slight clawing of the fingers and bending of the wrist being present. This gradually improved.[1]

Sub-divisions of the Motor Area.

In the area for face, head, arm, leg and trunk, the neurologist should be able to locate for the surgeon, through a study of motor phenomena, at least seven or eight different sub-areas; and in order to do this it is imperative for him to have exact knowledge not only of the anterior and posterior limits but also of the horizontal sub-divisions of this zone. Too much stress cannot be laid upon the proper separation of the region into horizontal levels; for, as Horsley[2] has well put the matter, the variation in the representation of motor function is greater in passing over the motor area from above downwards than from before backwards.

Horsley suggests the horizontal sub-division of the motor area mentally by means of certain sulci and their imaginary extensions. The imaginary extensions backwards of the superior and inferior frontal sulci through the central or Rolandic fissure subdivide with approximate accuracy the pre-central or ascending frontal gyre into three areas or zones from above downwards, namely, for the lower extremity, upper extremity, and face. According to Horsley, also, a line drawn forward from the anterior lower end of the intra-parietal sulcus will mark distinctly the division between the representation of movements of the upper limb and of the face behind the fissure of Rolando. This suggestion however is not as good a one practically as that with reference to the two frontal sulci. In the human brain at least the lower end of the intra-parietal sulcus is by no means fixed. It is often as low down as the end of the central fissure. It is better simply to place the posterior part of the area for the face in the lower third or fourth of the posterior central convolution.

The old method of subdividing the motor zone was by cutting the fissure of Rolando into thirds, and locating a circular or elliptical area over each of these thirds on both sides of the fissure—and upper area for the lower extremity, a middle one for the upper, and a lower one for the face. Such a subdivision is not now exact enough for accurate topographical diagnosis for operative purposes.

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[1] January 3, 1889, it is reported to me that this patient has had no spasms since the operation. He has regained almost entirely the use of his right hand and arm. He is still aphasic, although he has acquired the use of a few words.

[2] *Am. Jour. Med. Sci.*, April, 1887, p. 342-369.

Instead of subdividing the central or Rolandic fissure into thirds, it is better, perhaps to divide it into fourths, placing the area of representation for the lower extremity in the first fourth; that of the face in the lower fourth, and the areas for the upper extremity include the second and third fourths. This makes the diagrammatic method of representation correspond more closely to the results of recent investigations, as the vertical extent of the arm region on the lateral aspect of the hemisphere is about twice as great as that for the leg and somewhat greater than that for the face. The fissure of Rolando does not extend usually as far as the Sylvian fissure, and therefore making the junction of the third and last fourths of the former fissure the upper boundary of the face area, gives this area a greater height than that for the leg, but not as great as that for the arm.

Although time will not permit lengthy consideration, it will be interesting briefly to discuss some of the ascertained facts with reference to the sub-areas or centres in this wonderful motor region.

Subdivisions of the Face Area.

In the first place, the face area is best subdivided into an upper and a lower sub-area. In the upper sub-area movement of the opposite angle of the mouth and of the lower face generally are represented. In three cases during operations on human beings I have observed faradisation of the anterior superior portion of this face area produce contraction of the opposite angle of the mouth and face. It is probable that in the extreme upper anterior portion of this area immediately adjoining the area for the head and eyes, is a sub-centre for such movements of the upper face as contraction of the frontalis and orbicularis palpebrarum muscles. Such a centre does not come out clearly as the result either of physiological investigation or the experiments of disease, because associated movements are apt to remain even after destruction of a centre for such movements on one side of the brain. Of the face area Horsley and Schäfer say that it is physiologically remarkable that many of the movements which result from its excitation are apt to be executed bilaterally, which is only exceptionally the case with excitation of the other areas (except that of the head and eyes). Excitation of the upper third or half of the area, they continue, causes winking or closure of the eyelids, elevation of the ala of the nose, and retraction and elevation of the angle of the mouth.

An observation of Dr. Berkeley of Baltimore helps to fix with positiveness the exact location of the cortical centre for the movements of the angle of the mouth, chiefly performed by the zygomatic muscles, in this upper anterior portion of the general area for the face at a point about opposite the usual position of the fissure between the middle and inferior frontal convolutions. Gowers, in his 'Manual of Diseases of the Nervous System,' page 663, has a cut from a photograph furnished by Dr. Berkeley, showing a small focus of softening in the ascending frontal convolution at this point. This very circumscribed focal lesion caused persistent clonic spasm, chiefly of the zygomatics. My observation on the case trephined by Dr. Hearn confirms this position for the centre for this movement, as gentle faradisation of the cortex at this spot caused distinct drawing of the mouth upwards and outwards. The centre for the orbicularis palpebrarum, as stated, is doubtless in close proximity, probably just above the position of the centre for the angle of the mouth. While it is not unusually the case, paralysis in the upper distribution of the facial nerve sometimes does take place as the result of cortical lesion. In one of my cases of tumour in the posterior portion of the second frontal convolution, and causing some destruction by the invasion of the ascending frontal, distinct lagophthalmus of the opposite side was present. No lesion of the cranial nerves at the base was present. In this case ptosis was present on the other side, that is, on the side of the lesion. The fact that in facial spasm whether secondary after a peripheral facial paralysis or primary from nerve or central lesion, the orbicularis palpebrarum and zygomatic muscles usually act together so that the eye is closed or partly closed at the same time that the angle of the mouth is drawn upward and the naso-labial furrough deepened, is clinical evidence in favour of the close proximity of the zygomatic and orbicularis palpebrarum centres in the cortex. Close examination of cases of hemiplegia and monoplegia will often show weakness of the movement of closure of the eyelids in the paralysed side, a paresis which would not be observed by a careless observer.

The lower two-thirds of the face-area may be divided into at least two parts, an interior and a posterior. As the question of the exact function of this lower anterior portion of the face area is one of considerable importance, and one about which some doubts still exist, I will briefly detail some of the facts with reference to this sub-centre. Beginning with the most recent contribution to the question, it may be first stated that Dr. Felix Semon working in conjunction with Professor Horsley,[1] found that the lower end of the ascending frontal gyrus anteriorly is also excitable, an effect being produced upon the glottis by its excitation, viz.: to bring about phonatory closure of the vocal cords.

A fair amount of other evidence has been collected to show the existence of a cortical centre for laryngeal movements. In 1877, Seguin[2] reported a case of left hemiparesis without loss of consciousness but with impairment of speech, and also of phonation, the patient after the attack never being able to control the pitch of the voice, apparently from a lack of proper action of the muscles of the pharynx and larynx. Autopsy showed the surface of the right third frontal convolution degenerated, being yellow, tough, and elastic. The same change to a less extent was found in the same location on the left side. From such observations a motor centre for laryngeal movements had been sought for in the posterior extremity of the *right* third frontal (homologous with the speech centre on the left side, in right-handed persons).

Krause,[3] in the laboratory of Professor Munk, in 1883, investigated this question experimentally. On excitation of the cortex he noticed rise of the larynx and movement of the vocal bands to a position midway between expiration and phonation, lifting of the palate, contraction of the constrictor pharyngis, and movements at the base of the tongue. With extirpation experiments he found that eight dogs had lost the power of barking, on attempting which they uttered only a hoarse whine or made a sound.

Delavan[4] in 1865 contributed a paper on laryngeal cortical centres, in which he records some valuable facts and refers to the observations of Seguin, Krause, and others.

Garel[5] read before the French Society of Otology and Laryngology, in April 1886 an interesting communication on the laryngeal cortical centre, and vocal paralysis of cerebral origin, in which he reported a case with the details of an autopsy and a

[1] Phil. Trans. Royal Soc., vol. clxxix., 1888.

[2] Referred to by Delavan in *Med. Rec.*, N.Y., Feb. 14, 1885.

[3] Cited by Horsley and Schäfer and Delavan.

[4] *Med. Rec.*, N. Y., Feb. 14, 1885.

[5] '*Annales des Maladies de l'Oreille et du Larynx.*' Tome xii., 1886, p. 218.

sketch of the locality of the lesion. The inferior portion of the precentral gyre on the *right* side was slightly adherent to the meninges. The membranes being stripped the surface beneath presented a light yellow discolouration. At the foot of the third frontal gyre were two points of red softening, but there was no lesion of the anterior portion of the third frontal. On section these lesions were found to involve only the cortical substance, at the upper part only very slightly invading the white. The lesion of the precentral penetrated slightly into the white substance. It would seem from these experiments and observations that a centre for the movements of the larynx and throat is in the extreme lower anterior portion of the precentral convolution, and that it is probably better differentiated in the right than in the left hemisphere.

Somewhat numerous pathological observations corroborate the existence of Ferrier's oro-lingual centres also in the lower the anterior portion of the face area, probably a little behind the centres for the throat and larynx. In these oro-lingual centres are located particularly the representation of the movements produced by the orbicularis oris, and of protrusion of the tongue. Recently a case of typical oro-lingual paresis with involvement of this region has been observed by me, some details of which will be given later when speaking of the alleged sensory functions of the motor cortex. Pathological observations also somewhat numerous have confirmed the position of Ferrier's centre for movements performed by the platysma myoides muscle in the face area behind the Rolandic fissure.

In the hinder lower portion of the face area is probably represented opening and shutting movements of the mouth and retraction of the tongue.

Intra-cerebral Facial Tracts.

The existence and location of separate intra-cerebral facial tracts is a subject bearing a direct relation to that of the cortical areas and sub-areas for the face, and also a matter about which our knowledge is scanty.

Kirchoff[1] in 1881 reported the case of a man aged twenty-four, who had several attacks in which he became giddy, had convulsive tremors, lost power of speech, was unable to swallow, had profuse salivation, and drawing of his face to the left. Examination showed that he articulated with difficulty; labials and gutturals especially were troublesome; linguals he spoke with comparative ease. The lips were moved little in speaking; he could not whistle, but was able to approximate the lips. Saliva flowed from the mouth and there was excessive secretion of tears. The tongue was not protrusible more than one centimetre from the mouth, and it moved clumsily in the act of biting. At the time of examination swallowing was unimpeded, but the glottis was closed tardily. The patient often laughed without occasion. There was disease of the mitral valve. Ten days before death his face was suddenly drawn to the right and his left arm and leg became powerless. Convulsions occurred from time to time up to his death.

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[1] *Archiv. f. Psych.* Bd. xi., and *Brain*, July, 1881.

The *post-mortem* revealed embolic softening of the posterior two-thirds of the right corpus striatum (caudate nucleus), the underlying internal capsule, the outer segment of the lenticular nucleus, the claustrum, external capsule and island of Reil. The focus of softening in the lenticular nucleus was distinguished from the other softened portions by being surrounded by a wall of compact sclerosed tissue. Careful microscopic examination failed to show any disease of the medulla or pons. The author attributes the glosso-labial paralysis to the lesion of the lenticular nucleus, and the hemiplegia to the quite recent lesion of the caudate nucleus, internal capsule, and other parts. Cases of bilateral affection of the face, tongue, and throat, caused by unilateral lesion of the cerebrum, are rare; the author cites two, recorded by Lepine and Magnus respectively.

Ross[1] also reported a case of brain disease simulating bulbar paralysis in which the lesions were cerebral, in the ganglia and alongside of them in the capsules. In 1880 I observed a third similar case at the Philadelphia Hospital.

Hobson[2] in 1882 reported a case without autopsy—the main symptoms being left hemiparesis or paralysis, or paralysis of the tongue, difficulty of deglutition, speechlessness, clenching of the jaws; the patient had one inarticulate sound for everything, and a slight sound on laughing. In 1882 Ross[3] in an interesting paper on labio-glosso-pharyngeal paralysis of cerebral origin, traversed the literature of the subject, giving also some interesting original observations.

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[1] 'Diseases of the Nervous System,' vol. ii.
[2] *Brit. Med. Journ.*, April 29, 1882.
[3] *Brain*, July, 1882.

Subdivisions of the Arm Area.

The subdivision of the area for the upper limb, according to Horsley, is for the shoulder in the upper part, the elbow next below and behind, the wrist next below and in front, the thumb lowest and behind. In the area just above the superior frontal sulcus the movements of the lower and upper limb are absolutely blended, most markedly in the hinder sixth of the superior frontal gyre. Sometimes an epileptic fit from a lesion centred here begins by complicated or generalised movements of both extremities on one side.

At various points on the posterior central convolution, Ferrier it will be remembered fixed centres, excitation of which caused flexion of the thumb and fingers and firm clenching of the fist, with the synergic action of the wrist and fingers, but he did not differentiate centres for different flexors and extensors. From my own observation, I believed that the centres for movements of extension of the fingers and wrist are a little anterior and below those for flexion of the same parts.

In one of Keen's operations[1] the position of the hand centre was fixed by means of the faradic current. The fissure of Rolando was determined by both the methods of Hare and Thane. The trials with the faradic currents were made according to the determination of Dr. Keen, on both the post-Rolandic (post-central or ascending parietal) and pre-Rolandic (pre-central or ascending frontal) convolutions, and also the posterior extremity of the second frontal convolution. Excitation of the post-Rolandic convolution produced no effect. On touching the cortex with the electrodes at a position which apparently corresponded to the anterior portion of the pre-Rolandic convolution just back of the precentral fissure, movements of the wrist and fingers were produced. The hand moved in extension in the mid-line and to the ulnar side at different touches, the fingers being extended and separated. Above the region in which these movements were obtained, application of the current caused movement of the left elbow, both flexion and extension, and of the shoulder, which was raised and abducted. Below the region where the hand movements were excited the application of the current produced an upward movement of the whole of the left face. In the case of Hearn and the writer, reported above, the exact movements described by Keen were produced, that is, the extension at the wrist and separation of the fingers; also, below the spot where these movements were produced, an upward and outward movement of the face, or rather angle of the mouth. In our case however elbow and shoulder movements were not produced except as a secondary result. We obtained however a primary movement of flexion of the fingers and hand to which Keen does not refer, and which presumably was not produced. This movement resulted from touching with the electrodes a spot a little above and behind the place where the movements of the extensors were caused. Continuing and increasing the strength of the faradic applications at this point, flexions took place in succession of the fingers and thumb, and at the wrist and elbow. Keen estimated the portion of the convolution as containing the hand centre as about 1¼ inches long, and he places the centre for the wrist and fingers in the pre-Rolandic gyrus, its lower limit being at three-eighths of an inch above the temporal ridge, and its upper end where it fused with that of the elbow thirty-two millimetres higher up. The shoulder he placed still higher, while the centre for the upper face was in the same convolution below. These results correspond closely with those of Horsley. These facts of experiments on man would seem to uphold the view that the motor zone, in man at least, is much more extensive in front of than behind the fissure of Rolando. These results of Keen were all produced by excitation of the cortex anterior to the fissure of Rolando, supposing of course his determination of that fissure to have been correct. As nearly as I could determine the location of my own results were the same.[1]

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[1] Trans. of Am. Surg. Association, vol. vi., 1888, and also in the *Am. Jour. Med. Sci.*, November, 1888.

I was present at the operation in the case of Lloyd and Deaver.[2] By following Reid's and Horsley's lines, an area was exposed which was supposed to be on both sides of the Rolandic fissure, about the junction of the middle and lower third of the central convolutions. The faradic current was then used to identify locations. When the electrodes were applied to a point which was supposed to be just back of the fissure of Rolando, the

[1] October 4th, 1888, since the meeting of the Congress, Nancrede of Philadelphia, before excision of the cortex, fixed the position of the thumb centres by means of the faradic current. The patient suffered from convulsions which began with strong flexion of the right thumb, followed by extension of the wrist and fingers, pronation of the forearm and hand, flexion at the elbow, powerful flexion and rotation of the head to the right, thrusting out of the tongue between rigid jaws, and coincident with all conjugate ocular deviation to the right. The spot at which the faradic current was applied was estimated by Nancrede, to be from below upwards, in the second fourth of the ascending parietal convolution.—*Medical News*, November 24, 1888.

[2] Since reported in the *Am. Jour. Med. Sci.*, November, 1888.

movements which occurred were in order turning of the thumb on the palm, flexion of the fingers, flexion of the wrist extending to flexion of the elbow. At a point in front and below faradic stimulation caused marked contraction of the face muscles of the opposite side. "The mouth began to contract and was drawn to the left with a tremulous motion, and soon the tongue began to protrude toward the left corner of the mouth. Soon the left thumb began to be contracted and adducted into the palm; then the fingers contracted into the palm, and about the same time the face muscles began to contract more actively, while the head was drawn to the left, and the left eyelid began to work. At the same time the hand was gradually closed, and contraction of the forearm and arm began, while the latter was drawn from the side to an angle of forty-five degrees (deltoid action), and contractions of the biceps occurred. At no time in the course of these faradic applications, anywhere within the area exposed by the trephine and forceps, did any contraction of the leg muscles occur."

Subdivisions of the Leg Area and of the Trunk Area.

The movements of the lower extremity are represented in the upper portion of the motor area, and the adjoining marginal convolution; probably hip and thigh movements on the lateral and mesial aspects of the hemisphere near the median fissure, well formed in the area, and movements of the leg and toes farther back on the lateral aspect and also in the para-central lobule, and marginal convolutions of the median surface.

A narrow strip of the anterior portion of the leg area appears both from the results of experimentation and of pathological and surgical observation to be a trunk area, this being larger proportionally on the mesial than on the lateral aspect of the hemisphere, as represented in the diagrams. Horsley and Schäfer occasionally obtained movements of the trunk when the electrodes were applied to the lateral surface near the margin of the hemisphere. On the adjoining mesial surface, however, excitation produced rotation and arching of the lower spine and the pelvis, and extension of the hip, movement of the tail to the opposite side, and flexion at the knee.

Horsley[1] says that at the summit of the ascending frontal gyrus begins the representation of the lower limb only, the primary movement being that of the hallux. He describes a

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[1] *Am. Jour. Med. Sci.*, April, 1887.

case of traumatic epilepsy, the primary movement consisting of flexion of the hallux followed by the gradual flexion of the rest of the lower limb, and that followed by successive invasion of the rest of the lower body in the usual order. A dense and cystic cicatrix was found at the upper end of the ascending frontal gyrus. In another case, in which a tumour was removed and with it the cortex in front of the upper end of the fissure of Rolando, the only permanent complete paralysis of the lower limb was that of the hallux. In one of my own cases a small gumma involved the upper fourth of the ascending frontal and a smaller segment of the ascending parietal, crossing the upper extremity of the Rolandic fissure. This patient had severe attacks of left-sided spasm, beginning with twitchings in the left toe and foot; she also had partial paralysis of the left leg and arm, most marked in the leg. The leg area however as shown by Horsley and Schäfer, is largely situated upon the mesial surface of the hemisphere. According to these authors, the excitation takes effect chiefly upon the ankles and digits, producing most commonly flexion of the foot with flexion of the digits. The most marked movement in front of the upper end of the Rolandic fissure is flexion of the leg at the knee, with the addition, when the electrodes are applied more anteriorly, of flexion at the hip.

Area for the Movements of the Head and Eyes.

I cannot agree with Seguin in the recent paper by Weir and Seguin[1] that the centre for ocular movements is quite certainly not in the second frontal gyre as claimed by Ferrier and Horsley. Much is in favour of the view that it is situated in this neighbourhood.

In one of the cases of Horsley, in which operation was performed at the point of the meeting of the areas for the movement of the trunk, protrusion of the upper limb, and turning the head and eyes, the aura was contraction of the abdominal muscles followed by turning of the head and eyes to the opposite side. Other cases have been reported in which turning of the head was the starting point of the spasm. In some cases at least, when the aura or signal symptom can be most certainly shown to be the turning of the head and eyes to the side opposite to the supposed site of the lesion, the probabilities are that the focus or primary seat of the irritation is from a lesion in this oculo-motor region. The fact that cortical oculo-

[1] *Op. cit.*

motor palsies are not present as a persistent condition even when we have definite lesions of the second frontal gyre is not an argument of weight against the existence here of oculo-motor centres. Such persistent oculo-motor paralysis was not present in one of the best defined cases of lesions in the second frontal gyre ever reported, a case occurring in my wards at the Philadelphia Hospital. Such symptoms do not persist because of the automatic nuclear mechanism of the cranial nerves related to these centres.

In the last edition of Ferrier's 'Functions of the Brain,' he adheres to his views as to the position of the oculo-motor centres, and gives some new experiments bearing upon the subject. Irritation of the base of the superior and middle frontal convolutions in monkeys gives rise to lateral movements to the opposite sides with dilatation of pupils. The expression assumed by the animal is that of attention or surprise. The same movement, however as Ferrier himself states, also occurs along with other special reactions on stimulation of the angular gyrus and superior temporo-sphenoidal convolution more especially. With the latter is associated pricking of the ear from stimulation of the auditory centre. Ferrier argues that although the effects are the same the causes are different. Stimulation of visual and auditory centres attracts attention movements, the same as would result from stimulation of the motor centres for those movements. Destruction of the oculo-motor centres of Ferrier, according to some experiments, causes conjugate deviation towards the side of the lesion. Bilateral destruction of these centres for the first day caused inability to turn the head and eyes, but the animal recovered. Horsley and Schafer, and Ferrier also, got no motor or sensory symptoms from lesion of the pre-frontal lobes, except in one case in which the paralysis of the lateral movements of the eyes following the lesion of the post-frontal centres having completely disappeared, the destruction also of the pre-frontal regions caused rapid oscillations of the head, apparent inability to turn the head except *en masse* with the trunk, and drooping of the right eyelid. These facts, according to Ferrier, show that the pre-frontal regions belong to the same centres as the post-frontal, just as the occipital lobes belong to the visual centres.

Attempts have been made to remove the post-frontal as well as pre-frontal region. The animal could not maintain the upright position or move its head or eyes laterally. The eyes were kept shut except on cutaneous or other sensory stimulation. Some microscopical examinations of degenerations of tracts seem also to prove that the post-frontal regions contain the oculo-motor centres. Descending sclerosis from the innermost or mesial bundles of the internal capsule does not extend below the pons, but probably into the oculo-motor nuclei.

Horsley believes that the focus of representation of the movement of the turning of the head and eyes to the opposite side is in the middle frontal gyre, but also that these movements have a much more extensive representation.

"It must be left, for the present," he says, "an open question as to how far the representation of this important and interesting conjugate movement extends *forward* in the frontal lobe. A definite answer can only be given when the homologies between the sulci and the frontal lobe in the Macaque monkey and man have been thoroughly determined. That this area of function is continued over the margin of the hemisphere into the marginal convolution, has already been shown by Professor Schafer and myself. * * *

"In every instance the head and neck are turned to the opposite side, and in some parts there is produced at the same time or later, conjugate deviation of the eyes."

Horsley and Schafer in their contribution to the 'Philosophical Transactions' speak as follows with reference to this area: "The *head area or area for visual direction* comprises an oblong portion of the surface of the frontal lobe, extending from the margin of the hemisphere, round which it dips for a short distance, outward and somewhat backward to the upper and anterior limit of the face area. Posteriorly, it is bounded by the arm area, and in front by the non-excitable portion of the lobe. It extends therefore in front as far as the extremity of the precentral sulcus, and it includes the middle part of the frontal lobe above the antero-posterior limb of the sulcus, the part included in the angle formed by the antero-posterior and vertical limbs of the sulcus, and perhaps a small portion of the ascending frontal gyrus, close to the vertical limb of the same fissure. The effects produced by excitation of this are similar to those described by Ferrier as resulting from excitation of the rather more limited area marked 12 in his diagrams, viz.: opening of the eyes, dilatation of the pupils, and turning of the head to the opposite side, with conjugate deviation of the eyes to that side. If the electrodes are applied near the angle of the precentral sulcus, the ears are frequently also retracted."

In the patient referred to in whose case trephining was performed a weak current applied forward of the position at which movements of the fingers and hand produced, caused distinct deviation of the head to the opposite side. As nearly as could be determined the electrodes were applied over the extreme posterior portion of the second frontal gyre.

Conjugate deviation of the head and eyes, when a persistent or permanent symptom, is most likely to arise from lesions of the pons, cerebellum or cerebellar peduncles.

I have thus tried to indicate the recognised centres and subcentres of the motor zone. To such great works as that of Ferrier on the 'Functions of the Brain,' and to such monographs as those of Horsley and Schäfer, Horsley and Beevor, and Seguin, I must refer those especially interested in obtaining fuller details.

Overlapping Areas.

Some of these areas it will be seen apparently overlap each other, so far as their cortical representation is concerned, hence giving positions for trephining in some cases over the border of two adjoining areas. It might be said that with a large trephine it will not be necessary to separate and localise so many areas, as an opening 1½ inch or 2 inches can be made, and even this can be enlarged by the rongeur until a suspected lesion is reached, but this is a crude method in these days of precision. Even in cases of comparatively large lesion, the complete success of the operation will depend somewhat upon the first position in which the opening is made. The ideal position would of course be one that corresponded to the centre of the lesion.

Wonderful indeed is this motor zone of the cerebrum, a marvellous mosiac of centres of function, wrought from the great conceptions and priceless labours of the artists of our own guild; a mosaic, to each block, angle and jointure of which the neurologist can point the surgeon and say, cut here or there, or touch not this or that.

Different Classes of Localising Symptoms; their Characteristics and Comparative Value.

The neurological diagnostician must make use of his knowledge of these areas after a definite plan if he wishes to turn it to the best account.

When localising lesions he must go beyond even the important distinction advanced by Brown-Sequard, and very properly insisted upon and elaborated by all subsequent writers upon localisation, namely, the differentiation between symptoms of irritation and those of destruction. He should appreciate the possibility of six classes of symptoms presenting themselves for his consideration, namely, those of (1) local irritation, (2) local destruction, (3) local pressure, (4) invasions by lesions growing from adjacent areas to those under determination, (5) local instability, (6) reflex action at a distance.

In this connection I will only treat broadly of a few points, as the necessity for this subdivision of symptomatology becomes apparent when considering localisation in special regions. In the motor zone the symptom of irritation is especially spasm; but irritation symptoms may occur in other localities. In the visual, aural, olfactory, gustatory, or cutaneous areas they may take the form of hallucinations or other perversions of the senses. Symptoms indicating destruction are, in the motor areas, paresis or paralysis, and in other regions such manifestations as hemianopsia, word or mind blindness, word deafness, anæsthesia, analgesia, anosmia, &c. Pressure and invasion symptoms may, of course, be indicative of irritation or destruction, but are considered by the clinician in their relations to special areas under process of determination. Invasion symptoms will at first commonly be phenomena of irritation, and later both of irritation and destruction. By symptoms of instability I refer to those manifestations which occur as the result of discharging cortical areas without demonstrable gross lesions. Symptoms of reflex action will occur mostly in connection with lesions of the cranial or other nerves, and of the cerebral membranes, particularly the dura mater. They will receive particular attention when discussing some of the sources of error in motor localisation.

Certain characteristics, both general and special, of cortical spasm should be well understood. These have been best studied by a few observers, such as François-Franck and Horsley. In Franck's great work the peculiarities both of cortical, sub-cortical and capsular spasm have been determined by electrical experimentation, and are carefully described and graphically represented, the phenomena having been enregistered. Horsley, practically concurring with Franck, enumerates these characteristics as the presence of a period of latency, then tonic spasm, then clonic spasm, arrest of respiration with cyanosis and salivation.

A study of the initial symptom or sign in a case of irritative cerebral lesion, and also of the serial order of phenomena, may be of the utmost importance. Seguin has proposed to call this initial symptom the "signal symptom." Horsley's view of the manner in which movements are represented in the motor cortex is that in any given part of the cortex as minute as can be examined experimentally, there is represented a definite movement or combination of movements, being the primary movement and elicited by minimal stimulation only; and that secondary movements are due to the subsequent invasion by the discharge of nerve energy of portions of the cortex which lie nearest to and are in close relation with the parts stimulated. The primary movement gives the signal symptom of Seguin, and the secondary movements represent the "serial order" of phenomena.

The signal symptom in Jacksonian spasm has already been made use of in a number of cases to guide the surgeon in part or whole in selecting the site for operation.

In one of Horsley's cases there was first tonic extension and clonic spasm of the right lower limb. "The right upper limb was then slowly extended at right angles to the body, the wrist and fingers being flexed; the fingers next became extended, and the clonic spasms of flexion and extension affected the whole limb, the elbow being gradually flexed. At this time, spasms in the lower limbs having ceased, those in the upper limb continued vigorously. The spasm gradually affected the right angle of the mouth, spreading over the right side of the face, and followed by turning of the head and eyes to the right."

In another case first came "clonic spasmodic opposition of the left thumb and forefinger. The wrist next, and then the elbow and shoulder were flexed clonically, then the face twitched and the patient lost consciousness. The hands and eyes then turned to the left, and the left lower limb was drawn up. The right lower limb was now attacked, and finally the right upper limbs. Paralysis of the left upper limb frequently followed a fit. At frequent intervals every day the patient's thumb would commence twitching, but the progress of the convulsion could often be arrested by stretching the thumb and applying a ligature."

In another case by the same surgeon the spasm was ushered in with a desire to defecate, sometimes with sharp pain in the left side of the belly. Then followed tightness of the throat, and sometimes spasmodic cough. Then the head and often the eyes turned to the right; the right arm was jerkily protruded, and the patient became unconscious. All the limbs became powerfully flexed, as a rule, but the lower limbs were frequently extended.

Weir and Seguin, Keen, Lloyd, and Deaver, the writer, and others have taken advantage of the signal or initial symptom in fixing a site for operations, and thus, either with or without gross lesion, hand centres, thumb centres, face centres, &c., have been excised.

Even movements of the trunk have been used to guide opera-

BRAIN.

OCTOBER, 1889.

Original Articles.

ORIGIN OF HUMAN FACULTY.

BY GEORGE J. ROMANES, F.R.S.

HAVING been requested by the Council of the Neurological Society to read a paper on a recently published book of my own, for the purpose of raising a discussion on the psychological doctrines which are therein presented, I will begin by briefly stating the aim and scope of the book in question.

The title of the book is "Mental Evolution in Man;" but as the work constitutes only the first member of a series which I intend to devote to this topic, its second or subsidiary title more accurately defines the limits of its subject-matter —namely, "The Origin of Human Faculty." The aim of this treatise is twofold. First, to meet upon their own ground those various writers—psychological and theological —who maintain that a great exception must be made in the case of the human mind to the otherwise uniform law of continuous evolution; and, secondly, to indicate the probable causes, and thus to trace the probable history, of the transition between the intelligence of the lower animals and the intelligence of man.

It appears to me that before the Neurological Society I may be allowed to adopt the first of these positions without argument, and will, therefore, assume that in some way or another the transition in question has taken place. On the basis of this assumption I shall be free to devote all the time at my disposal to a consideration of the probable causes, or method, of the transition. For this purpose it is needful to set out with a brief analysis of ideation.

If I look at any particular face now before me, I receive what is called a perception, or a percept, of that face. If I then close my eyes, or turn them away from that face, but still retain the memory of it before what Hamlet calls "the mind's eye," I have what is designated an image or an idea of the face which I had previously perceived. The idea which I should have in this case would be what Locke calls a Simple Idea—that is to say, the idea of a particular object, or the mere memory of a particular percept. But now suppose that before shutting my eyes I had taken a general survey of all the faces at present before me, I should then have what Locke calls a Compound Idea, or the idea of a face in general, as distinguished from my previous simple idea, or the idea of a single face in particular. It is of great importance to note that these compound ideas are created by a fusion of a number of individual percepts, and thus differ from simple ideas in that they are something more than the mere memories of particular percepts. It is needless to say that animals possess compound ideas as well as simple ideas. For instance, a dog has a compound idea of Man, as distinguished from his particular idea of Master. But, lastly, when we come to what Locke calls General or Abstract Ideas, we find, as he says, "that which puts a perfect distinction betwixt man and brutes." Wherein, then, consists the difference between a compound idea and a general idea? It consists, according to the unanimous agreement of nearly all writers, in the idea having been named by a word, or other sign, which is designedly used as the mark or symbol of that idea. For instance, like my dog, I have a compound idea of Man, and a simple idea of some particular man; but, unlike my dog, I can name the one by the general word Man, and the other by the particular word John. A compound idea, when thus named, becomes what is called a conception, or a concept. Now, it will be observed that this conceptual order of ideation differs entirely from the other two orders which we have just been considering, in that a symbol is substituted for the mental image, so that the symbol may be used instead of the image, whether or not the image is present to the mind—or, indeed, whether or not any equivalent image admits of being formed at all. Consequently, the mind is now enabled to deal with symbols of ideas without requiring to call up the ideas themselves as memories of perceptions. Consequently, also, the mind is thus enabled to quit the sphere of sense and rise to that of what is called abstraction; furnished with the wings of language, human thought can soar far beyond the possibilities of any ideas which could be suggested by merely sensuous experience.

It will be further observed that the psychological condition to thus naming ideas, so as intentionally to treat the names as symbols of the ideas—the psychological condition required for this is the presence of what is called Self-consciousness. Unless an agent is conscious of itself as a mental agent, and of its own ideas as ideas, it is clearly not in a position to bestow upon them names as names. The mind must be able, so to speak, to get outside of itself, in order to contemplate its own states as such, before it can name these states with the conscious intention of using the names as symbols. In other words, the mind must be capable of introspection; and this power of introspection it is that goes to constitute the one and only distinction between the human mind and mind of lower orders, whether we call this distinction the faculty of Self-consciousness, of Abstraction, of Reason, of Logos, or by any of the other terms which are habitually used to signify this unique power of a mind to turn in upon its own self and examine its own ideas.

Thus far psychologists of every school are agreed. But as a great deal of laxity has been displayed by responsible writers in the use of Locke's terms, and, moreover, as his intermediate division of compound ideas has been largely lost sight of, I have devised for this intermediate division what I think are more appropriate terms, viz., *Generic Ideas* or *Recepts*. Adopting, then, these terms, you will note that all ideas admit of being classified under one or other of three divisions—viz., Simple Ideas, Generic Ideas, and General Ideas; or, more briefly, Percepts, Recepts, and Concepts. Percepts and recepts are common to the lower animals and to man; but concepts belong to man alone. Moreover, while recepts are formed by an automatic fusion of percepts, without any intentional activity on the part of the mind itself, concepts can only be formed by the intentional activity of the mind in the act of naming a percept or a recept, for the purposes of symbolic abstraction. Thus, a recept is passively received into the mind, while a concept is actively conceived by it. For example, observation shows that water-fowl have one recept (or organised body of percepts) answering to water, and another recept answering to land. So has man. But, unlike the fowl, he is able to bestow on each of these recepts a name, and so to raise them both to the level of concepts. Now, in order to do this, he must be able to set his recept before his own mind as an object of his own thought; before he can bestow his conceptual names on these ideas, he must have cognised them as ideas. In virtue of this act of cognition, he has created for himself—and for purposes other than locomotion—a priceless possession; he has formed a concept.

Nevertheless, the concept which he has thus formed is an exceedingly simple one—amounting, in fact, to nothing more than the naming of some among the most habitual of his recepts, "land" and "water." But it belongs to the nature of concepts that, when thus formed, they admit of being intentionally compared and grouped together into higher and higher concepts, which, in virtue of being successively named, become further and further removed from the sphere of sensuous perceptions. Thus there arises a kind of algebra of recepts. Now, it is in this algebra of the imagination that all the higher work of ideation is accomplished; and throughout it depends on the power of a mind to contemplate its own ideas as such.

The difference between a mind which is capable only of receptual ideation, and a mind which is capable, even in the lowest degree, of conceptual ideation, is usually taken to depend on the absence in the one and the presence in the other of the faculty of Language. Therefore, it is here necessary to say a few words upon this subject.

The faculty of language is, in the largest signification of the term, the faculty of making signs. Now, there is no doubt that the lower animals present the germ of this faculty. A dog will bark significantly before a closed door as a sign to request that it shall be opened; a wise cat will pull one by one's clothes as a sign to come to her kittens if they are in danger; a parrot will depress its head as a sign to be scratched, and so forth. Nay, a parrot will even use verbal signs with a correct appreciation of their meanings, as proper names, substantives, adjectives, and verbs.[1] Where, then, is the difference between this kind of sign-making, which we may call receptual sign-making, and the sign-making which is peculiar to man, and which alone is conceptual sign-making? The difference is broad and deep. It consists in the power which the human mind displays, as already explained, not only of naming its ideas, but of making one idea stand before another as itself an object of thought. In other words, a man is able to think about his own ideas as ideas. Not only, like a parrot, can he name a particular man John (in consequence of having heard that particular man called John, and therefore associating the name with the man), but he is able to think about this name as a name. And similarly, in all other cases, the difference between naming a thing receptually by mere association, and naming a thing conceptually by intentional thought, is all the difference between knowing that thing and knowing that we know it. And the difference on the side of the talking or sign-making agent, is all the difference between an agent that is conscious only, and an agent that is likewise self-conscious. For it is the faculty of self-consciousness which thus enables a mind to set one idea before another as an object of its own thought; by means of this faculty the mind is able, as it were, to stand outside of itself, and so to perceive objectively the ideas which are passing subjectively—and this just as independently as if it were regarding an external series of dissolving views. How it is that such a state of matters is possible, whereby a mind can thus, as it were, get outside of its own existence, and so regard its own ideas as objective to itself—this is the mystery of all mysteries, the bottomless abyss of personality. But, accepting the fact as a fact, all that we have at present to do is to note the enormous difference which the presence of this fact introduces with reference to the sign-making faculty. For it means that merely conscious or receptual sign-making is sign-making which is not thought about as such; while self-conscious or conceptual sign-making is sign-making that is thought about as such. Consequently, while a parrot can only learn words or phrases which are stereotyped in the frame-work of special associations, man, after having thus learnt his vocabulary, can afterwards use his words and phrases like moveable types, whereby to convey any number of different meanings by changes of their relative positions. Thus there are names and names; names receptual and names conceptual. In short, it is his super-added faculty of self-consciousness that has made man *par excellence* the sign-making animal; and therefore what we have to do to-night is to consider the genesis of this faculty.

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[1] All such statements on matters of fact, here and elsewhere, rest upon evidence which is furnished in my book.

First of all, however, I should like to say something more about the sign-making faculty, as this occurs before the rise of self-consciousness—that is to say, in the brute and in the human infant.

I distinguish four grades of the sign-making faculty. First there is what may be called the *indicative stage.* Long before it can speak, the infant will express its simple desires by means of intentionally significant tones and gesture-signs, such as pointing to objects in connection with which it desires something to be done. Here the infant is obviously at the same level of sign-making as the cat which pulls one's dress to signify "come," or the parrot which will depress its head to signify its desire to be scratched.

Next we find what I call the *denotative stage* of sign-making. Here names are bestowed receptually, or by special association, upon particular objects, qualities, actions, and states of feeling. This stage occurs in the child when it is first emerging from infancy, and is psychologically indistinguishable from that which obtains in the talking birds. Denotative names, then, are names which have been learnt by merely receptual association; they do not imply any self-conscious or conceptual thought.

Following upon the denotative stage is what I call the *connotative.* This consists in a receptual extension of the meaning of a name from the thing which was at first denoted by that name, to other things which are seen to resemble it. Thus, for example, as M. Taine has remarked, a young child which has learnt the name *Bow-wow* for a house terrier, will soon extend it to all other dogs, then to pictures of dogs, to images of dogs, to his elder brother when walking on hands and knees, and so on through ever-widening circles of connotative extension. Now I have observed that a parrot will do precisely the same. One of the birds which I kept under observation, used to bark in imitation of a terrier in the same house. Soon the barking became the parrot's denotative name for the terrier, so that the bird would bark whenever it saw the terrier. After a time it ceased to do this, but would always bark when it saw any other dog. Thus the parrot resembled the child of which M. Taine speaks, in that it extended the significance of its name for a particular dog, so as to apply it to any other dog. Here, however, the connotative extension of the name ceased; the bird would not bark at pictures of dogs, no doubt because it was not intelligent enough to perceive the pictorial representations.

Lastly, there is what I call the *denominative stage* of sign-making, or the bestowal of a name consciously known as such. Here we arrive at what I mean by conceptual naming, and therefore this stage of sign-making cannot arise until the mind has attained to self-consciousness. Therefore, also, it only occurs in man, and first appears in the growing child between the second and third years. Then, of course, the child begins to predicate, or to arrange its names in the form of propositions.

Now, in connection with our subject, it is of the highest importance to note, not only that the three first stages of the sign-making faculty are thus common to animals and human beings, but also that these three first stages advance very much further in the growing child than they ever do in any animal, even before the growing child attains to the fourth, or distinctively human stage. In other words, even while still moving in the purely receptual sphere, the growing child becomes much more intelligent, and much more proficient in the art of making signs, than any animal. Although not yet a self-conscious agent, and therefore not yet having attained to conceptual thought, a child between two and three years of age has already distanced every animal in respect of its purely receptual intelligence. But observe, thus far no difference of kind can be alleged by our opponents, because to allege any difference of kind between one order of receptual intelligence and another, would be to vacate their whole argument. This argument depends on the distinction between ideation as receptual and conceptual —or between an agent that is, and an agent that is not, self-conscious. But a child up to its third year is not a self-conscious agent. This is proved by the fact that it never employs words having any self-conscious implication, and never gives evidence of even in the lowest degree thinking about its own ideas as such. In short, it cannot be disputed that the respects in which the intelligence of a child between two and three years of age distances that of the most intelligent animal, have reference only to a higher advance of receptual ideation; the ideation has not yet become conceptual, and therefore cannot be alleged by our opponents to differ from the ideation of an animal in kind. The higher degree of intelligence which is displayed by a child of this age must therefore be taken to consist in a higher development of receptual intelligence, just in the same way as a dog is more intelligent than a bird. In order to distinguish this higher degree of receptual intelligence, which only occurs in man, and in the growing child immediately precedes the first appearance of conceptual intelligence, I will call it *pre-conceptual* intelligence.

It is of importance to note how far this higher receptual, or pre-conceptual intelligence, can go, and therefore I will briefly consider the kind of language or sign-making (*a*) which leads up to it, and (*b*) by which it is expressed when attained.

The indicative stage of language in the infant is at first below that of the more intelligent animals. But very soon it becomes equal to that of the most intelligent. The child will then point to objects in connection with which it desires something to be done, in just the same way as a dog will beg before a water-jug, &c. It will pull one's dress in the same way as a cat does to signify "Come;" and, lastly, it will use its voice to make significant—though inarticulate—sounds, after the manner of all the more intelligent of the higher animals. Thus far, then, the child is still moving in the same levels of receptual ideation as the higher animals. But very soon its receptual ideation begins to distance that of even the most intelligent animal: the ideation of the child has therefore entered upon what I call its pre-conceptual phase. From this point onward its gesture signs become correspondingly more and more significant, so that in children who are late in beginning to talk, it may develop into regular pantomime. But now note, it is impossible that as yet there can be any conceptual ideation, because as yet there are no names, and therefore an absence of so much as the condition to the performance of any act of introspective thought.

Thus much, then, for the indicative phase of language in the receptual and pre-conceptual levels of human ideation. Passing on now to the next, or denotative phase (which the indicative phase may largely overlap in children who are late in talking), we find that when a child first begins to use articulate signs, it learns the use of them in just the same way as a parrot does; that is to say, it learns the name of particular objects, qualities, actions, and states, by special association—in other words, receptually. So far, then, as the beginning of the denotative stage of language is concerned, there is no difference at all between the child and the parrot. Neither is there any difference with regard to the beginning of the connotative stage; for, as I have already said, a parrot will extend its denotative name for a particular dog to all other dogs the resemblance of which one to another it is able to perceive—just in the same way as a young child will extend its name of *bow-wow* from a terrier to a mastiff. And although the bird will not follow the child where the child takes the further step of extending the name from living dogs to pictures of dogs, this is plainly due to the intelligence of the bird not advancing far enough to perceive the resemblance of pictures to the objects which they are intended to represent. Many dogs, however, and certain monkeys are able to do this, and, therefore, if a dog or a monkey were able to articulate, there can be no doubt that the brute would follow the child through this further step in the connotative extension of a name. Indeed when we remember the extraordinary degree in which monkeys are able to understand the meanings of words, as well as the extraordinary propensity which they show in the way of imitating the actions of mankind, there can be no question that if it were not for the anatomical accident of monkeys being unable to articulate, they would follow a child through what would probably seem a surprising distance in the use of denotative names and receptually connotative words. The chimpanzee now at the Zoological Gardens, which I have taught to count as far as five, displays in a perfectly marvellous degree the power of understanding language—so that one can explain to her verbally what one wishes her to do, in just the same way as we explain this to an infant of about eighteen months old. Therefore, if this animal had been able to articulate, there can be no doubt that it would answer us in the same way that a child answers us when first emerging from infancy.

But here we come to an important point in our comparison between the two cases. After a child does emerge from infancy, its receptual intelligence continues to grow; and it continues to grow until it has left far behind the receptual intelligence of any brute. That is to say, between the time that a child first parts company with the brute in the matter of sign-making, up to the time when it first begins to use denominative words, or words which are used with a true conceptual appreciation of their significance, there is an immensely large interval which is filled by advancing stages of receptual development. Before it has attained to even the earliest dawn of self-consciousness—and therefore before it has attained to the possibility of thinking about names as names, or of ideas as ideas—the child has made a prodigious advance in its receptual intelligence, and therefore in the sign-making whereby this intelligence expresses itself. Now, as already stated, in order to distinguish this large and important territory of ideation, which is occupied by the mind of a child between the time that its receptual intelligence parts company with that of the most intelligent animal, up to the time when it first reaches the truly conceptual or self-conscious intelligence of a human being, I call this intervening territory of ideation by the name pre-conceptual. Pre-conceptual ideation, then, is that order of higher receptual ideation which is not presented by any brute, but which is presented by the growing child between the time that its developing intelligence parts company with that of even the most intelligent animal, up to the time when the dawn of self-consciousness begins to convert this higher receptual ideation into ideation that is truly conceptual.

I will now briefly consider the kind of sign-making which is distinctive of this pre-conceptual stage of ideation. The child has now acquired a large number of denotative words, which it has learnt by special association to regard as significant of certain objects, qualities, actions, and states. Suppose, then, that it sees its little sister crying. Its denotative name for this sister is *Dit:* its denotative name for the action of crying is *Ki.* Now the object and the action which these two names severally denotate happen to occur together before the child's observation; by the mere force of special association, therefore, the child denotates them both simultaneously—that is to say, brings them into *apposition.* This apposition in consciousness of two habitual recepts with their corresponding denotations is thus effected *for* the child by what may be termed "the logic of events:" it is not effected *by* the child in the way of any intentional or self-conscious grouping of its ideas, such as goes to constitute the distinguishing feature of the logic of concepts. Therefore, when on seeing its sister crying, the child says, *Dit Ki,* although in one sense we may say that the child is making a proposition, in another and a truer sense we must deny that is making a proposition. The proposition is what may be called pre-conceptual, not conceptual: it is of the psychological kind that we might have expected a monkey to make, if a monkey had been able to pronounce denotative names as well as it can understand them. For the proposition is made by an agent which is not yet a self-conscious agent, and therefore cannot possibly have been thought about as a proposition. That is to say, it lacks the very element of conceptual or introspective thought on which our opponents rely as proving a difference of kind between the brute and the man. Therefore, without argumentative suicide, our opponents cannot afford to maintain that a pre-conceptual proposition of this kind is a genuine proposition, in the sense of being a proposition that implies for its construction any of the distinctively human powers of introspective or abstract thought.

Now, it is needless to say that at this age a child is incessantly making these pre-conceptual propositions; and, of course, the important thing to notice about them is that as yet they are not, and cannot possibly be, conceptual propositions. It is not until the child has attained to self-consciousness, and therefore is able, not only to denotate, but to denominate—not only to name, but to think the names, not only to make statements, but to contemplate its statements as such—it is not until the child has taken this further step that it has the peculiar quality of ideation on which our opponents rely for their psychological distinction between the brute and the man No doubt these pre-conceptual propositions are strongly suggestive of a near approach to true or conceptual propositions: but the point is that as yet they do not present the very feature which it is necessary that they should present, if they are to conform to the distinction of kind between animal and human intelligence which our opponents have endeavoured to institute. They are always evoked by the external logic of events bringing into apposition objects, qualities, &c., the denotative names of which are called up in the child's mind by immediate association—and, therefore, are necessarily called up in apposition. Thus the apposition which here gives to the

going on in other minds; and with that kind of recognition of self as an active and suffering agent to which allusion has just been made. But, over and above the animal, the child has now at its command a much more improved machinery of sign-making, which, as we have before seen, is due to the higher evolution of its receptual ideation. Now, among the contents of this ideation is a better apprehension of the mental states of other human beings, together with a greatly increased power of denotative utterance, whereby the child is able to name receptually such mental states on the part of others as it thus receptually apprehends. These, therefore severally receive their appropriate denotations, and so gain clearness and precision as images of the corresponding states experienced by the child itself. "Mama pleased to Dodo," would have no meaning as spoken by a child, unless the child knew from its own feelings what is the state of mind which it thus attributes to another. Hence, we find that at the same age the child will also say, "Dodo pleased to mama." Now, it is evident that we are here approaching the very borders of true or conceptual self-consciousness. The child, no doubt, is still speaking of itself in objective phraseology; but it has advanced so far in the interpretation of his own states of mind as clearly to name them, in the same way as he would name any external objects of sense-perception. Thus, he is enabled to fix t [illegible] tates befor
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of continuity among its own states wherein the full introspective consciousness of self consists.

In confirmation of this my general argument, I must now conclude by observing that, although the advance to true self-consciousness from lower grades of mental development is no doubt a very great and important matter, still it is not so great and important in comparison with what this development is afterwards destined to become, as to make us feel that it constitutes any distinction *sui generis*—or even, perhaps, the principal distinction—between the man and the brute. For even when self-consciousness does arise, and has become fairly well developed, the powers of the human mind are still in an almost infantile condition. In other words, the first genesis of true self-consciousness marks a comparatively low level in the evolution of the human mind—as we might expect that it should, if its genesis depends upon, and therefore lies so near to, those precedent conditions in merely animal psychology to which I have assigned it. But, if so, does it not follow that, great as the importance of self-consciousness afterwards proves to be in the development of distinctively human ideation, in itself, or in its first beginning, it does not betoken any very perceptible advance upon those powers of pre-conceptual ideation which it immediately follows? There is hus show [illegible] even less reason for regarding the first [illegible] ual self-consciousness as marking a [illegible] of kind, than there would be so to [illegible] ose higher powers of conceptual [illegible] tly—though as gradually—super[illegible] od and youth. Yet no one has [illegible] that the intelligence of a child [illegible] display a difference of kind.

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although isolated and imperfect, this source of evidence is one of immense importance—standing, in fact, to the science of comparative psychology in very much the same relation as palæontology stands to the science of comparative anatomy; since it serves, by a kind of fossil record, to mark a prehistoric development of ideation, which is curiously analogous to the geological record of a prehistoric development of organisation. Moreover, the evidence thus furnished is of special value on account of its wholly independent character; it is throughout perfectly distinct from the psychological analysis on which we have hitherto been engaged. Doubtless you will already have perceived to what it is that I allude: it is to the independent, and I venture to add, the overpowering, witness of Philology. The gradual evolution of articulate language has preserved for us a kind of palæontological record of the gradual evolution of conceptual thought, with the result of showing that in the life-history of the human species, as in the life-history of the individual child, this conceptual thought derived its origin from these pre-conceptual levels of ideation which have already been occupying our attention. Although it is impossible for me now to give even an outline sketch of this argument from philology, I may conclude by quoting the last paragraph of my summary, in order to give you a general idea of the immense assistance which is thus rendered to the theory of evolution in the domain of human psychology.

"Here, then, I bring to a close this brief and imperfect rendering of the 'Witness of Philology.' But brief and imperfect as the rendering is, I am honestly unable to see how it is conceivable that the witness itself could have been more uniform as to its testimony, or more multifarious as to its facts—more consistent, more complete, or more altogethe overwhelming than we have found it to be. In almost every single respect it has corroborated the results of our psychological analysis. It has come forward like a living thing, which, in the very voice of Language itself, directly and circumstantially narrates to us the actual history of a process the consistent steps of which we had previously inferred. It has told us of a time when as yet mankind were altogether speechless, and able to communicate with one another only by means of gesticulation and grimace. It has to us described the first articulate sounds in the form of sentence-words, without significance apart from the pointings by which they were accompanied. It has revealed the gradual differentiation of such a protoplasmic form of language into 'parts of speech;' and declared that these grammatical structures were originally the offspring of gesture-signs. More particularly, it has shown that in the earliest phases of articulate utterance, pronominal elements, and even predicative words, were used in the impersonal manner which belongs to a hitherto undeveloped form of self-consciousness—primitive man, like a young child, having therefore spoken of his own personality in objective terminology. It has taught us to find in the body of every conceptual term a pre-conceptual core; so that, as the learned and thoughtful Garnett says, '*nihil in oratione quod non prius in sensu*' may now be regarded as an incontrovertible axiom. It has minutely described the whole of that wonderful aftergrowth of articulate utterance, through many lines of divergent evolution, in virtue of which all nations of the earth are now in possession, in one degree or another, of the god-like attributes of reason and of speech. Truly, as Archdeacon Farrar says, 'to the flippant and the ignorant, how ridiculous is the apparent inadequacy of the origin to produce such a result.' But here, as elsewhere, it is the method of evolution to bring to nought the things that are mighty by the things that are of no reputation; and when we feel disposed to boast ourselves in that we alone may claim the Logos, should we not do well to pause and remember in what it was that this our high prerogative arose? "So hat auch keine Sprache ein abstractum, zu dem sie nicht durch Ton und Gefuhl gelangt wäre." To my mind it is simply inconceivable that any stronger proof of mental evolution could be furnished, than is furnished in this one great fact, by the whole warp and woof of the thousand dialects of every pattern which are now spread over the surface of the globe. We cannot speak to each other in any tongue without declaring the pre-conceptual derivation of our speech; we cannot so much as discuss the 'origin of human faculty' itself, without announcing, in the very medium of our discussion, what that origin has been. It is to Language that my opponents have appealed; by Language they are hopelessly condemned."

A
CONTRIBUTION TO THE PATHOLOGY OF NIGHT:
NOCTURNAL PARALYSIS.

BY CH. FÉRÉ.
Médecin de Bicêtre.

THE influence of night upon animals and on man in health and in sickness has attracted the attention of many investigators. It has been attributed to various factors —sleep, cold, hygrometric condition of the atmosphere, darkness, inactivity—each of which, without doubt, plays a certain *rôle*, but this *rôle* is still badly defined.

In discussing this question there are certain physiological conditions of sleep which deserve consideration. Boussingault shewed that a turtle dove burned 255 milligrams of carbon per hour when awake, but that when asleep the consumption fell to 162 milligrams. Scharling says that the amounts of carbon consumed by a man asleep and awake vary as to 1 to 1·237. During the night also the respiratory movements are less energetic and slower (Becquerel) and the rate of pulsation diminishes.[1] One is compelled to admit, however, that the physiological conditions of sleep are very complex and difficult to isolate experimentally.

Darkness of itself exercises a marked influence on the vital functions. Moleschott has shown that the quantity of carbon dioxide exhaled in the dark is to the quantity exhaled in the light as three to five, and that the proportion varies with the intensity of the light. Bidder and Schmidt have shown that among animals in a state of inanition the loss of weight due to the exhalation of carbon dioxide and to transpiration tends to become equal for day and night when the animals are made blind. The influence of white or coloured

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light on animal nutrition and development has been demonstrated during the past few years by the works of Moleschott and Fubini,[2] Uskoff,[3] Gysi and Luchsinger,[4] Schenk,[5] Pouchet,[6] Platten,[7] &c. In addition Edwards had already shown the influence of light on the development of batrachian larvas. The relation established by Darwin between the colour of flowers and the fertilisation of plants by certain insects shows in another way the influence of coloured rays upon animals. Nor has this influence been observed by naturalists only, for "a Mr. de B. declared that his manner of conversation with Madame De—— had changed since she had altered the colour of the furniture in the room from blue to crimson" (Chamfort). Moreover an attempt has been made to utilise the action of the various colours in the treatment of mental diseases. I have shown that in certain subjects this excitatory action may make itself apparent by modifications of the muscular force, of the circulation, or of sensibility, the excitatory value diminishing, as a rule, from red to violet.[8] The action of luminous excitation on muscular energy has been confirmed by the experiments of M. Charpentier on the muscles of the eye.[9] The result of the absence of luminous excitation on the energy of muscular efforts had not previously escaped observation altogether, as Reydelet[10] mentions that night marches are the more fatiguing, and recalls an observation of von Humboldt concerning a Countess of Madrid, who lost her voice at sunset and recovered it at dawn. The effects of sensory excitation, and more especially of visual excitation, which I had recorded,[11] have been also confirmed by the experiments of M. Urbantchich.[12] The influence of luminous excitation on the sensibility of the other sense organs is moreover illustrated by a fact of common experience. Many smokers have remarked that in the dark they cannot tell so well, either by taste or smell, if their cigar be lighted or not.

The diminution of sensibility under the influence of darkness had not completely escaped observation. Taillefer[13] noticed that during the night impressions are received more slowly and are more fleeting. It is easy to verify experimentally the retardation of the reaction when the eyes are closed. Occasionally I have found an increase of time amounting to seven or eight-hundredths of a second in people perfectly healthy. I have observed that with some hysterical people the effect of darkness exhibits itself by a diminution in the volume of the upper limbs—easy to see with a pletismograph—by an increase of electric resistance, by hemato-spectroscopic modifications,[14] by diminution of the amplitude of respiratory movement,[15] &c.

Many of the lower animals unprovided with eyes still manifest an evident sensibility to light. The observations of Tremblay on hydra, of Pouchet on fly larvas, and of Plateau on blind myriapods, leave no doubt on this head. This dermatoptic function evidently exists to a certain degree in man, and with certain subjects it would probably be sufficient to try opportunely, in order to render it apparent. I have seen in many hysterical persons the modifications of the volume of the hand and of the muscular force that one provokes by occlusion of the eyes notably diminished when a large expanse of skin was exposed to the light.

The lowering of external temperature which occurs during the last hours of the night has also a great influence on vital, and more particularly nervous, phenomena. Helmholtz having determined the rapidity of the nervous current in the nerves of a frog at twenty-six metres per second, found that this rapidity may be reduced to fifteen metres when the temperature is lowered to 0°C. The lowering of temperature affects not only the physiological activity but also the nutrition of the nerves after they have been cut. Among frogs, too, segmentation, which requires only fifteen to twenty days in summer, does not appear till the third month in winter; Vulpian and Philipeaux found that the excitability persisted for six months in winter. Heat, on the contrary, favours in general the fulfilment of nervous functions, and also of the psychical ones.[16]

Modifications of the hygrometric conditions of the atmosphere during the night add their own effects to those produced by temperature. Barral has studied to what extent the humidity of the air causes diminution of the quantity of water exhaled.

The electric condition is connected with the hygrometric state of the atmosphere. During the night atmospheric electricity finds in the humidity of the air a good conductor to or from the earth, as the case may be.

All these conditions favour a falling off of nutrition during the night both among animals and plants. Whatever may be the relative importance of these different factors, their collective influence asserts itself by diminution of the intensity of nutrition during the night—a diminution which entails a lowering of temperature in the organism. Von Boerensprung, Gierse, Ladame, Ogle, Jurgensen, &c., have shown that the lowest temperature occurs from four to seven o'clock in the morning. Women, whose normal combustion appears less intense (Hirn), are more sensitive to these cosmic influences, particularly to the lowering of temperature (Gavarret). This influence of the night, and of the absence of excitation, is evidenced by the physiological conditions of hibernating animals. Marshall Hall[17] had already noticed these conditions, and described the effects of the gentlest excitation of the so kindred animals. The falling away of the nutrition among them is so great that Schiff has observed that in a marmot the atrophy of the inferior end of the crural nerve cut five weeks before was not more advanced than the nerve of a dog after five days. In some individuals affected by a great depression of the nervous system one may observe sometimes during the night such functional modifications that one can truly say they are subject to a veritable nightly hibernation. One has attributed to the night a special influence upon certain physiological acts, such as childbirth, which appears in fact more frequent during the night, and also natural death. This influence remains unexplained, but I may add, as far as natural death is concerned, that the tolerably numerous data which I have been able to bring together give no evidence of the influence of the night in this respect.[18]

Although the night has an evident influence on a considerable number of physiological phenomena, it has a still greater one on the phenomena of disease. Sometimes this influence appears determinative, at other times only exaggerative. A large number of painful diseases present nocturnal exacerbations. The usual hours for osteocope pains are well known. Neuralgia, and articular pains frequently intensify during the night. Gouty attacks in particular often occur towards two o'clock in the morning, when the atmospheric temperature is approaching its minimum. Wilks[19] says that it is at this same hour in which nutrition becomes very low that certain ailments of the stomach, hemoptysis, more especially show themselves, and that among epidemics the cholera commences its attack. In fact, it was ascertained at Munich that out of a hundred cases seventy had begun in the night time. The sweats of phthisical patients, which are more of the nature of paralytic exudations than of active secretions, occur by preference during the night towards early morning. A considerable number of cases of dyspnœa become worse during the night. In spasmodic asthma the attacks generally appear during the night, and relief can occasionally be obtained by the action of a bright light. Laennec has recorded a case of this kind in which the attacks were rendered less severe when the lamp was lit. Epileptic fits often increase in frequency towards the morning,[20] but this increase may be variously interpreted.

The influence of the night was well known to the ancients. Homer calls it the ruler of men and gods. Hesiod describes it as bringing forth all the mischief-making spirits of darkness, and makes it the goddess of misfortune; but what had struck them most of all was the influence it exercised over the mental state. It was the great nurse of sorrows—*nutrix maxima curarum.* (Ovid.)

Night plays a considerable *rôle* in the appearance and development of delirium,[21] and this influence in not due to sleep and dreams[22] alone. M. Baillarger remarked long ago[23] that with some insane people lowering of the eyelids was sufficient to provoke optical hallucinations. Allison[24] has recorded certain nocturnal mental troubles occurring exclusively among overworked men of business.

Nocturnal terrors, according to the note of M. Debacker,[25] occur occasionally in over-worked individuals, among whom they form a sort of delirium. In studying nocturnal terrors it is advantageous to distinguish two orders of facts. These terrors are most frequently the effect of terrifying hallucinations, which arise during sleep, and persist for some time on awaking, sometimes, indeed, till they are dispelled by the appearance of the light. In certain cases, however, nocturnal terror occurs without reference to any fixed mental representation; it then consists of a feeling of pain akin to that of agarophobia, sometimes, indeed, so intense that one might compare it with angina pectoris, and is caused solely by the deprivation of light, occasionally even in the open day in darkness artificially obtained. It is said that Hobbes was incapable of supporting the absence of light. I have already mentioned the case of an individual who, surprised by darkness in going through a tunnel in an unlighted carriage, experienced a sensation of choking and of oppression on the chest, along with a pain so severe that had he not been caught he would have fallen by the door. Another person in the same circumstances fell in a veritable collapse, with incontinence of urine and fæces. Among many neuropathic people the effect of darkness manifests itself only by melancholic and hypochondriacal tendencies which show themselves at nightfall. Besides, it is well known that many invalids experience an increase of their troubles and fancies at this part of the day, and like to ask for more attention: "the night leaves all its powers to grief and only weakens reason." (de Staël.)

Vesanic suicides often occur in the morning, and the influence of nocturnal depression on thoughts of destruction is clearly shown by the fact that they frequently disappear after the patient has partaken of any stimulant whatever, or even of some light food. This circumstance may be cited in passing as a support for the hypothesis of the organic origin of pessimism which I have elsewhere sustained.

The most characteristic troubles, however, relate to the sensory and motor functions. The influence of the night can manifest itself by indispositions which may moreover be connected with another cause. The powerlessness of drunkards, for example, is obviously increased by the night.

In the morning they experience a general lassitude, or are in a paretic state which frequently affects the lower limbs, sometimes also the member which plays the greatest *rôle* in the exercise of their profession. Under the influence of a painful emotion the paralysis may become complete.[26] The morning impotence of drunkards shows itself particularly in the exaggeration of the trembling movement, which diminishes in consequence of new excitations.

The various impotences which can be properly connected with the influence of the night present a combination of ailments both motor and sensory. The first which may have attracted attention is the incontinence of urine. Of this series J. L. Petit recognises three groups according to their cause, viz., the incontinence of children too lazy to get out of bed, the incontinence of those who sleep so soundly that they are not awakened by the sensations of distension, and the incontinence of those who believe they are relieving themselves somewhere. It is but just, however, to recognise with Trousseau that the first cause of incontinence is a neuropathic predisposition,[27] but the classification of J. L. Petit does not the less merit consideration in so far as it takes account both of motor and sensory ailments, the existence of which is incontestable. The indecision and dislike of movement which causes children to lie in bed until the contractibility of the bladder overcomes the resistance of the sphincter muscle, scarcely differs from the indecision and failure of the will that one observes among many neuropathics under the influence of the night. Besides, it is probable that the sphincter muscle, which is so sensible to peripheral excitation and to emotions of every kind, loses some of its tonicity when luminous excitation is wanting, and that all things being equal it does not resist so well simply because of the darkness. This explanation is by no means theoretical, as the experiments of Mosso and Pellacani have shewn that the bladder is sensitive to all kinds of excitation; and, on the other hand, I have been able to show that in man the energy of the sphincter of the anus, which is functionally analogous to the sphincter of the bladder, undergoes notable modifications under the influence of sensory, and more especially luminous, excitation.[28] At the same time Mosso and Pellacani admit that in darkness the sensation of distension is weakened.

Another nocturnal syndrome, in which sensory troubles combine with motor ones, is well known under the name of hemeralopia or nocturnal blindness. Although this blindness does not, like nocturnal incontinence, occur exclusively among neuropathics afflicted with congenital nervous exhaustibility,[29] it nevertheless prevails among subjects who have undergone great exhaustion from different causes: (1) or general, as the consequence of acute disease (Gubler), in puerperality (Demeulater), in malaria, as the result of bad hygienic conditions in prisons, on board ship, in the army, in houses of education, where epidemics usually spare the better nourished and better lodged, (2) or local, in consequence of fatigue of the eyes in equatorial seas, or in snow fields. Sanson and Sichel hold that hemeralopia induced by these causes is an insensibility consequent upon too strong stimulation, and comparable with the deafness of men who work in the midst of intense noise. One might, however, with more justice compare it to the transitory anæsthesia which succeeds to the sensory paroxysms of epilepsy, and on which A. Hughes Bennett has recently insisted.[30] These forms of anæsthesia by exhaustion are not rare in hysteria, and I have already shown that they may be sometimes induced experimentally by making the patient wear red spectacles for a few minutes only. One may with perfect right connect these phenomena with motor paralysis from exhaustion, of which I have already had occasion to cite some examples,[31] and of which Mr. Suckling has recently discovered a case.[32] Hemeralopia is not only constituted by a periodic anæsthesia, but in a number of cases it is accompanied by sensorial disorders, such as mydriasis, paresis of the pupil, difficulties of accommodation, diplopia and even strabismus. In fits of nocturnal blindness, in fact, there occurs a series of phenomena quite the inverse of those which appear under the influence of a moderate increase of luminous excitation, which determines an abnormal activity of the special sensibility, and at the same time of the muscular appendages of the eye. One may therefore say that if hemeralopia be a syndrome due to general or local exhaustion, its attacks are produced under the influence of an insufficiency of the physiological excitant.

Paresis for want of physiological excitation occurs frequently among hysterical people under the form of a general depression of all the functions. On awaking, the patient remains in a condition of general torpor, incapable of movement, accompanied by general insensibility, and occasionally by a remarkable coldness of the skin. In many such cases I have ascertained that the contraction of the field of vision, the diminution in the acuteness of sight, and the chromatic sensibility were much more pronounced a short time after the patient got up than they were a few hours later. The result on nervous exhaustibility of the absence of excitation and of the modifications in nutrition which occur during the night, seem to me to be of interest in the interpretation of other phenomena which are much more frequent than one might expect.

Mr. Weir Mitchell[33] has recorded under the name night palsy or nocturnal hemiplegia cases of paralysis, showing itself usually on awaking, sometimes accompanied by a painful numbness and extending sometimes to the two hands, sometimes over the whole side of the body. This occurrence is most frequent among women at the period of the menopause, but occurs also among men.

Mr. Ormerod has described analogous cases, accompanied by swelling of the hands, in which he was particularly struck by the numbness, and the pricking occasionally severe enough to awake the patient. This numbness and paresis usually came on during the night, but might be induced during the day by certain exertions on the part of the patient, such as washing, rubbing or working with the needle. This last circumstance appears to me to be worthy of special note, for it seems to show in cases of "exhaustibility," where there is a question of prolonged exercise, that fatigue may have the same value as the want of excitation, and the lessening of nutrition which occur during the night. In the cases of Mr. Ormerod[34] which refer for the most part to women at the period of the menopause, the paretic numbness usually disappeared by simple friction.

Mr. Sinkler[35] has also observed this numbness, especially in women at the menopause. He considers that a certain *rôle* is played by overwork, and thinks that the troubles are due to hyperæmia of the nerve trunks or of the spine, favoured by nocturnal decubitus. These patients were cured by galvanisation of the spine, massage, and ergot of rye. Mr. Saundby[36] who has published similar cases with painful numbness, which he compares to that which follows compression of a nerve, accompanied by a blue coloration and coldness of the hands. He considers them to be a form of neurasthenia. The most serious cases he has met with were in men suffering from gastric troubles, who were cured by rhubarb and calomel. Replying to the note of Mr. Saundby, Mr. Notley[37] says he has seen the same disorders among women, and has treated them successfully with iron. He attributes them to anæmia. Mr. Moir,[38] on the other hand, has encountered them only in women at the menopause, usually dyspeptic, who were cured by bromide of potassium. Mr. Steavenson[39] has also observed them in women at the critical age, and is inclined, like Weir Mitchell, to admit that hysteria plays a part. Lastly, Mr. A. H. Smith[40] has recently described similar cases, and suggests that the nocturnal lowering of the circulation plays a certain *rôle* in their production.

The works I have just cited show that a paresis with numbness of the extremities exists, which arises under the influence of the night, disappears temporarily under slight excitation and definitely under a tonic, and at the same time quieting treatment. This paresis, which generally appears in exhausted subjects, may also occur without the influence of the night as a consequence of constrained exercise. In such case we have to deal with a special series of complex symptoms, which differs from the increase of pains one observes during the night in certain neuralgic patients,[41] in certain forms of paræsthesia,[42] and in erythromelalgy.[43] These paretic states appear to be only exaggerations of the phenomena which occur in a normal condition under the influence of the night.

The more transitory and less accented troubles that one observes every day, so to speak, in a great number of neurasthenics, and particularly in hysterical people, form a sort of transition between normal phenomena of life and those paretic states. As Weir Mitchell has fully recognised, hysteria seems to play an important rôle in the etiology of these diseases.

The two following cases may be useful as illustrations:—

CASE I.—Mrs. V. came to consult me for the first time at the Saltpetrière on the 12th January, 1885. She was accompanied by her mother, who was over sixty years of age, but still very agile, and looked much younger than she really was. The mother had a painful ovarian spot with slight anæsthesia on the left side. Although the menses had ceased nine years previously, she had been subject to migraine, with attacks of melancholia and occasionally convulsive fits. The father, who had been a drunkard and profligate, had quitted the house twenty-five years before, and no one knew what had become of him. A brother of the father had died in prison when undergoing confinement for swindling. Mrs. V. had had two sisters born after her. The one had died of convulsions connected with teething at the age of eighteen months; the other died of convulsions when only six months old.

Mrs. V. had been a precocious child both physically and mentally. She had walked and spoken at a very early age, and had learned very rapidly at school. She has never had convulsions nor tic, but from the age of six has suffered from frequent migraine, followed by vomiting, and during her whole life her sleep has been troubled by nocturnal terrors and nightmare. Menses began when she was twelve. At the age of seventeen she had an attack of chorea in consequence of worry. This lasted three months, and chiefly affected the left side. At nineteen she was married, and had her first child, a boy, when twenty-three. This child died of convulsions on the eighth day. In the following year she was delivered of a child still-born. During her third pregnancy she had anorexia and vomiting, which ceased spontaneously in the fourth month. The child, a girl, was born at the proper time, and though she has suffered occasionally from convulsions has been otherwise very healthy. She is now seventeen.

Since her chorea Mrs. V. has always enjoyed good health, and has had no distinctive nervous outburst until about three years ago. At that time her husband died, and she suffered severe monetary losses. This induced insomnia, followed by loss of appetite and emaciation. The state of her affairs being improved by dint of hard work her health became pretty good. About two months ago she had a very abundant metrorrhage, the organic cause for which could not be ascertained. It has not occurred again but she has remained pale, and the menstrual flow has been slight but painful. A few days after this mishap she began to feel constrictive pains in the head extending over the whole of the cranium, but predominating in the postero-inferior region, which appeared moreover to be the seat of a constant pressure. From time to time she heard a cracking noise in the back part of the neck, which resounded in the occipital region of the skull. At nightfall she was seized by painful fancies, of ruin for her mother and her daughter, of illness for all her friends; at the same time she was a prey to unusual pusillanimity and indecision. Her sleep was disturbed by dreadful nightmares. She was widely awake towards six in the morning, but was incapable of making any movement. She suffered from distension of the bladder, but could not even think of getting up. Her limbs seemed numb to her and as if wrapped in cotton. She appeared to know the position of her extremities only, and it seemed to her as if the greater part of each limb was awanting, and her hands and feet had been brought up quite close to her body. This sensation is analogous to that experienced by amputated persons, who say they feel only the extremity of the absent limb. She could make no movement whatever until her mother entered the room. When daylight was admitted a sensation of numbness and pricking gradually appeared in the extremities of the fingers and toes. These sensations, occasionally very painful, preceded the return of ability to move. It was now, as a rule, about eight o'clock, and by this time the patient could get out of bed, maintain herself in a standing position, and make movements of the arms. The complicated movements of the fingers, however, remained almost impossible. She was thus incapable of fastening her clothes or of taking up a pin. When she had moved her arms and been rubbed for a few minutes her fingers became more supple. From the time she awoke until the nearly complete restoration of movement took in general about three hours. One day when left in the dark till about ten o'clock she was found in the same helpless condition. Movements of the head and neck and of articulation were not affected. On examination no modification of the external aspect of the limbs could be determined. She suffered pain in the region of the left ovary, and had slight anæsthesia on the same side. The contraction of the field of vision was tolerably extensive, and the patient was insensible to violet rays in the left eye. The left iris is of a deeper brown and the pupil not so large as on the other side. Under the influence of bitters, iron, bromide of potassium, and hydrotherapy, combined with static electricity, all these symptoms disappeared in the space of three weeks with the exception of the pain over the ovary and the hemianæsthesia.

CASE II.—Mrs. P——, fifty-two years old, does not acknowledge any antecedent hereditary neurotic tendency. Her mother was rheumatic and died of some form of heart disease. Her father died of cancer of the pylorus. Among her relatives there exists a certain number of arthritic symptoms. She herself has never been ill, but from the commencement of menstruation at the age of fifteen she has been subject to very violent migraine, which returns about every fifteen days. These migraines begin by a supra-orbital pain invariably seated on the left. Little by little this pain extends to the frontal region and is accompanied by an exquisite sensibility of the skin—a sensibility so great that the slightest contact even with the hair causes an insupportable burning sensation. When this pain has persisted from about half-an-hour to an hour the patient begins to perceive scintillations which she localises on the left of both fields of vision. These soon give place to a sort of luminous disc, whose borders are indistinct and seem to vibrate rapidly. This disc, situated towards the left, slowly enlarges and at the same time becomes dark in the centre. In a few minutes the disc has assumed the appearance of a toothed saw hollowed out on the half of its internal circumference, or of a half crown of fortification *à la Vauban*. This toothed wheel is the same colour as electric light, but from time to time red and blue points appear. It is in a state of very rapid vibration. In proportion as the wheel opens and enlarges the centre becomes quite dark and obscure. At the end of an hour the toothed wheel has so enlarged itself as to occupy the limits of the field of vision and forthwith disappears. The patient then declares that she can by this time see only the right half of objects placed in front of her, and that when she looks at people they appear to her to be cut exactly in two on

[29] WEIR MITCHELL, "Lectures on Diseases of the Nervous System, especially in Women." 2nd Edit., 1885, p. 102.

[30] A. HUGHES BENNETT, "On Excessive Sensory Cortical Discharges and their Effects."—The *Lancet*, 1889, pp. 619 and 672.

[31] CH. FÉRÉ, "On Paralysis by Exhaustion."—BRAIN, 1888.

[32] C. W. SUCKLING, "Exhaustion Paralysis."—*Lancet*, 1889, p. 578.

[33] WEIR MITCHELL, "Lectures on Diseases of Nervous System, &c.," p. 181.

[34] ORMEROD, "On Peculiar Numbness and Paresis of the Hand.—*St. Bartholomew's Hosp. Rep.*, 1883, vol. xix., p. 17.

[35] SINKLER, "On a Form of Numbness, chiefly of the Upper Extremities."—*New York Med. Journ.*, 1884, vol. xl., p. 107.

[36] R. SAUNDBY, "On a Special Form of Numbness of the Extremities.—*Lancet*, 1885, p. 422.

[37] NOTLEY.—*Lancet*, 1885, p. 548 and 643.

[38] MOIR.—*Lancet*, p. 595.

[39] STEAVENSON.—*The Practitioner*, 1886, vol. xxxvi., p. 410.

[40] A. H. SMITH, "Waking Numbness, a Heretofore Undescribed Neurosis.—*Amer. Journ. Med. Sc.*, 1887, vol. xciii., p. 410.

[41] HANDFIELD JONES, "Studies on Function of Nervous Disorders," 1870, p. 550.

[42] PUTNAM, "A Series of Cases of Paræsthesia, mainly of the Hands, of Periodical Recurrence, and possibly of Vaso-Motor Origin."—*Arch. of Med.*, New York, 1880, vol. iv., p. 147.

[43] DANA, "Acro-neurosis."—*New York Med. Record*, 1885, vol. xviii., p. 57.

[44] CH. FÉRÉ, "Note sur cas d'amaurose hydro-traumatique." Comptes Rendus, Soc. de Biologie, 1886, p. 178.

[45] CH. FÉRÉ, "Note sur des accidents produits par la lumière electrique." Comptes Rendus, Soc. Biol., 1889, p. 365.

[46] CH. FÉRÉ, "A Contribution to the Pathology of Dreams and of Hysterical Paralysis."—BRAIN, vol. ix., 1887, p. 488.

THE TREATMENT OF CERTAIN DISEASES OF THE SPINAL CORD BY MEANS OF SUSPENSION.

BY MOTSCHUTKOVSKY, M.D., OF ODESSA.

I FIRST noticed an elongation of the body when witnessing the suspension of patients for surgical purposes in the process of application of Sayre's jackets. Repeated measurements convinced me that this elongation varied from 2½ to 5 cms. and that it involved the spinal column. Two problems occurred to me in consequence of this observation.

(1) What change does take place in the relations between the spinal cord, its membranes and nerve roots, and the vertebral canal during the process of suspension?

(2) Can suspension have a beneficial influence in certain diseases of the spinal cord?

I shall describe the results of my researches in chronological order and begin with the second question.

The history of the first case in which I tried suspension is as follows:—

CASE I.—P. M., from Cherson, aged forty-nine years. At the age of twelve he suffered from sores in the axillary and inguinal regions. These sores (which have left no scars) were cured according to the patient's statement by the use of certain powders. M. is unmarried, has never been addicted to onanism, has not suffered from acquired syphilis, has not exceeded sexually nor in drink. He was for many years in the service of a jobmaster, and [illegible] several occasions was severely injured. About twenty-five [illegible] ago he fell off a horse for the first time and injured his back. [illegible] nsequence he was laid up at this hospital for about six [illegible] Five years after the first accident he again fell from a [illegible] his back; on this occasion he recovered in about two [illegible] ut any treatment. Finally in 1871 his horses ran [illegible] ell from the cart dislocating his left ankle. He was again treated at the Odessa "Town Hospital" and was well in three months. After leaving the hospital he began to suffer from severe shooting pains in the arms, legs, back and waist. These pains he considered to be rheumatic and attributed them to his having taken cold, as he was much exposed to the weather in the course of his work. Up to the year 1880 the pains were severe chiefly immediately before and during bad weather; at other times he felt pretty well In January 1880 his legs gradually got weak; afterwards his arms were similarly affected. He next noticed that both his arms and his legs were getting thin. The pains now became constant and intolerable, not allowing him to sleep. The patient became unable to walk towards the end of 1881. On admission into the Odessa "Town Hospital" on the 15th of February 1882 patient could stand only with difficulty, and staggered considerably even with his eyes open. As soon as his eyes were closed he fell. He had great difficulty in walking forwards, although supporting himself with the aid of a stick. His feet were raised very much at each step and then rapidly brought to the ground. He could walk backwards much more easily even without a support; however, after a few steps he used to fall on his back. M.'s height was 1 515 metres; he was fairly well built, and weighed 49·65 kilos. The muscles of both upper extremities were somewhat flabby and slightly atrophied about the palms. When the hands were extended the fingers had a bird's-claw appearance. The muscles of the leg, the extensors of the foot especially were also flabby and said to have become wasted. The toes dropped considerably. Dynamometer to right hand, 22 lbs.; left, 20 lbs.; right leg, 85 lbs; left, 82 lbs.

The spinal column was symmetrical and normally sensitive to percussion and pressure. All the joints were normal; mucous membranes vividly coloured; skin flabby, without scars or spots.

On closing his eyes patient could not co-ordinate the movements of his legs. Even under guidance of vision in an effort to touch some object with the toes his foot was carried considerably beyond the object aimed at. The same phenomenon though in a much less degree was noticeable in the movements of his fingers. Elbow and knee jerks were absent. Temperature normal. Skin always dry, but not scaly. There were several dark brown pigmented spots on the thighs, the marks of slight injuries.

The musculo-cutaneous pain reflexes were examined [by means of a special reflexometer contrived by Dr. Motschutkovsky] on 15th of April and their latent period was determined.

Great toe of right foot, latent period	1·460″
,, left ,, ,,	1·738″
Index finger of right hand ,,	0·898″
,, left ,, ,,	1·0008″

The time requisite for the central conduction of painful impressions (*i.e.*, the time elapsing between a reflex act and the perception of a painful sensation) was as follows:—

Great toe of right foot	0·767″
,, left ,,	0·951″
Index finger of right hand	0·371″
,, left ,,	0·492″

Sensibility to pain was lessened in the inferior extremities more than in the superior, and on the left side more than on the right. Tactile sensibility was also diminished but not to the same extent, Muscular sensibility, the muscular sense and the sensation of position, were weakened. Thermic sensibility was fairly good, the patient being able to distinguish a difference of 2° in temperatures ranging from 14° to 35° R.

The faradic excitability of the muscles was diminished. The nerves and muscles of the extremities did not respond readily to the galvanic current, though the A and K contractions followed normally in their respective order. The patient complained of formication in his legs, of a feeling of pricking in the ends of the fingers of both hands, of a constant sensation of cold in the legs and of severe shooting pains in the region of the sciatic nerves. The pains were more severe in the right leg than in the left; they were intensified chiefly by sudden barometric changes. Moreover the patient complained of girdle pains about the abdomen and chest and sometimes of pains in the region of the cervical spine, extending into the arms. Sexual functions abolished. Mental state undisturbed. Taste and smell normal, but hearing deficient since fifteen years. Patient could distinguish the ticking of a watch with his right ear at the distance of 8 cms., and of 5 cms. with his left. The tympanic membranes were capped and dull. The pupils were contracted, and reacted only slightly to light. Field of vision normal; acuity diminished. Discs sharply defined, somewhat glistening. Retinal vessels not fully distended. Palpebral conjunctiva vividly injected. Sclerotic vessels dilated.

Chest tympanitic on percussion; expiration prolonged.

Defæcation is normal but micturition infrequent; the patient has to strain before beginning to pass water. The urine yellow, phosphatic, but without albumen or sugar; sp. gr. 1021. The patient was suspended on the 24th of April 1882 for the first time. After the second suspension the sciatic pains ceased; the sensation of cold in the legs was diminished; stick no longer necessary to stand. After eighteen suspensions the patient with his eyes open walked very well, though with some jerking forwards of the feet. He can stand on one leg for two minutes and walk without a stick with the eyes closed. After the twenty-ninth suspension sensibility to pain was much improved; no more formication and pricking in the tips of the fingers. The sensation of cold has been replaced by a pleasant feeling of warmth. July 1st the musculo-cutaneous pain reflexes also show improvement.

Great toe of right foot, latent period	0·923″
,, left ,, ,,	1·102″
Index finger of right hand ,,	0·722″
,, ,, left ,, ,,	0·790″

The time requisite for the *central* conduction of the sensation of pain was—

For the great toe of right foot	0·449″
,, ,, left ,,	0·531″
Index finger of right hand	0·251″
,, ,, left ,,	0·370″

Knee jerks still absent. Dynamometer, right hand, 33lbs.; left, 28lbs.; right leg, 90lbs.; left, 86lbs.

By the 14th of November after seventy-seven suspensions the musculo-cutaneous pain reflexes showed:—

Great toe of right foot, latent period	0·741″
,, left ,, ,,	0·852″
Index finger of right hand ,,	0·369″
,, left ,, ,,	0·280″

The time requisite for the central conduction of the sensation of pain was—

For the great toe of right foot	0·320″
,, ,, left ,,	0·482″
For the index finger of the right hand	0·204″
,, ,, ,, left ,,	0·137″

By that time he had so much improved that, according to his own expression, it was "unnecessary" to feel any better. He walks a good deal about the garden; can walk half a verst (two-thirds of a-mile) without resting and without using a stick. He can go up a flight of steps also without resting. The legs are painless and stronger. There still are some dull girdle sensations and occasional headaches, the latter being in fact more marked since suspension. Knee jerk still absent, but more frequent and marked returns of sexual feelings. Dynamometer pressure increased: right hand, 58lbs.; left, 46lbs.; right leg, 97½lbs.; left, 89lbs. Weight of patient, 49·4 kilos. On the 15th of March 1883 the following facts were ascertained:—

Musculo-cutaneous "pain-reflexes," latent period—

Great toe of right foot	0·722″
,, left ,, ...	0·795″
Index finger of right hand	0·232″
,, left ,, ...	0·202″

The time requisite for the central conduction of the sensation of pain was—

Great toe of right foot stimulated	0·320″
,, left ,, ,, ...	0·490″
Index finger of right hand ,, ...	0·200″
,, left ,, ,, ...	0·151″

Dynamometer: right hand, 59lbs.; left, 57lbs.; right leg, 98lbs.; left, 92lbs. The suspensions had to be given up at intervals for a couple of weeks or more, owing to pains in head and spine. They were usually carried out every other day, lasting at the onset for from one to two minutes, later on from four to seven minutes each. After the twelfth suspension an ecchymosis occurred in the left sclerotic lasting about ten days.

The general results of ninety-seven suspensions were:— (1) complete disappearance of shooting pains; (2) diminution of ataxic gait; (3) considerable improvement in the delaying of the musculo-cutaneous "pain reflexes;" and (4) of the conduction of painful impressions; (5) disappearance of certain paræsthesiæ (sensation of cold, of pricking, formication, girdle pains); (6) increase of muscular force in the extremities; (7) improved equilibrium when standing or walking on closing the eyes; (8) improvement of sexual feelings; (9) returning perception of pain in the toes and improvement of tactile sensibility; (10) increased size of atrophied muscles. Throughout the period of six months while suspension was being carried on, no change was noticed in the (1) body weight; (2) knee jerk; (3) pupil and rest of visual apparatus.

Case 2.—N. T., aged forty-two years. Duration of illness, five years. From the age of nine up to the time when he was admitted to the hospital, patient worked in a tobacco factory. Eighteen years ago he had a chancre which healed in the course of a month. It was accompanied by two buboes in the left groin. They were opened and suppurated for about three months. At the same time he had an abscess in the right axilla and a sore throat; the latter was however not ulcerated. Five years after this a pink rash appeared on his chest, abdomen and limbs. This rash persisted for about three months. The first syphilitic manifestation was treated by means of mercurial pills, Zittman's decoction and iodide of potassium. The eruption disappeared without any treatment; no further signs of syphilis have since been observed. Twenty years ago N. T. gave up drinking, to which he had been addicted for about six years. Married about four years ago and has one child whose health is perfect. Masturbation between fourteen and seventeen years of age; since then venereal excesses especially from 1870 to 1874. He used to live in Roumania where he had no ague, but four attacks of a fever of a typhoid(?) type (at the ages of eight, eleven, sixteen and eighteen years.)

Present State.—Weakness of legs; unable to get up from the bed or to stand for a single minute even with assistance. Micturition frequent; bowels act only every four to five days; complete sexual impotence; severe shooting pains in the arms and legs; abdominal girdle pains; occasional cutting pains about navel, followed by vomiting or diarrhœa (gastric crises); cold creepy sensations in the legs. Patient weighs 51·5 kilos; slightly built and anæmic. Keeps lying on his back, occasionally sitting up for dinner. Bony framework, normal; spine, symmetrical, but sensitive to pressure and to percussion in the cervical region. No roughness of knee joints though the synovial membranes are somewhat full. No true atrophy though muscular system generally flabby. Dynamometer: right leg, 45, left, 43 lbs. Squeeze of right hand 36, left, 24 lbs. On attempting to walk in the normal position no loss of power, but inco-ordination is noticed, the heel striking the ground before the toes; foot lifted with effort and a slight jerk, brought down with a stamp of the heel. Inco-ordination is very marked in movements executed when lying down; when told, his eyes being closed, to touch one knee with the opposite foot, patient raises the latter with two or three sweeps of the leg in the air, and finally misses his aim. Romberg's symptom is well marked; no inco-ordination of arms, no tendon reactions, nor cremasteric and abdominal reflexes. The latent period of musculo-cutaneous "pain-reflexes" are delayed (as measured by my reflexometer):—

Right big toe	0·736″
Left ,, ...	0·699″
Right index finger	0·276″
Left ,, ,, ...	0·325″

Integuments flabby and wrinkled; palms and soles bright red; skin of limbs never sweats and is cold; hair began to fall off about a year ago; nails thick and clubbed. Pin prick distinctly felt over upper trunk, arms, and head. At a lower level, from Poupart's ligament in front and from third lumbar vertebra behind, sensibility to pain is diminished; soles anæsthetic to pin pricks. Examination with Weber's compasses gives similar results. Touch normal except slight alteration in the toes, where the contact of camel's hair brush is felt as a pricking or burning. Sensibility to moderate temperatures chiefly affected; thus 14° C. and 24° C. are not discriminated; between 30° C. and 42° C. differences of 2° to 3° C. are appreciated pretty correctly. The muscular sense is much diminished; muscular sensibility less so.

Neuro-muscular galvanic reactions normal; excitability of calf to Faradic current somewhat diminished. The patient complains of a cold heavy feeling, passing sometimes into formication, in both soles. Severe lightning pains, shooting from buttocks down into great toes. Occasional attacks of perforating pains in the middle third of the legs and in the shoulder joints, especially on the right side. They increase at night, and though unaffected by temperature, they become intolerable during changes from wet to fine weather, and strong N. or N.E. winds. Morphia alone controls these pains. Bowels act but every four or five days; stools scybalous; occasional colics and diarrhœa. Micturition strained and frequent (thirty to forty times daily); sexual sense in abeyance since one year; sleep good in the absence of pains. No optic changes; pupils small, react feebly to shading. The other organs of sense and the mental faculties are normal; memory somewhat impaired. No thoracic nor abdominal symptoms of any importance; quantity of indican in urine.

We diagnosed a sclerosis of the posterior columns of the spinal cord, the patient being in the paralytic stage of the disease. The first suspension was effected on the 12th of May 1882 and lasted two and a half minutes. After this the patient was suspended on alternate days, or at longer intervals, for periods gradually increased to ten minutes. Seventh suspension: patient is able to get out of bed without assistance. Eleventh: begins to walk. Sixteenth: able to sit down and get up from a chair without assistance of hands. Twenty-second: shooting pains begin to subside.

Latent period of musculo-cutaneous "pain-reflexes":—

Right big toe	0·707″

Left big toe	0·710″
Right index finger	0·280″
Left ,, ,,	0·322″

Dynamometer: right hand, 92 lbs.; left, 82 lbs. Right leg, 107 lbs.; left, 98 lbs.

After sixty-one suspensions erections occurred, and micturition was unstrained and less frequent. Bowels act daily. Indican diminishes. The pupils less contracted in the light, dilated more in the dark. Weight, 49 kilos. Perception of painful impressions increased, a slight prick being readily felt. No change in thermic and tactile sensibility. Also the muscles react to faradism. Pain reflexes after sixty-five suspensions :—

Right big toe, latent period	0·712″
Left ,, ,,	0·664″
Right index finger, latent period	0 278″
Left ,, ,,	0·320″

Summary of progress after eighty suspensions, spread over ten months: (1) No shooting pains; (2) Some locomotion restored, patient able to go quarter of a verst without resting; (3) Micturition normal; (4) Bowels more regular, hence (5) diminished indican; (6) No gastric crises; (7) Return of sexual feeling; (8) Muscular strength of limbs improved; (9) Perception to pain in legs improved; (10) Ataxia less; (11) Pupil action improved; (12) Stands better with closed eyes. The treatment had no effect (1) on the latent period of the musculo-cutaneous pain reflexes; (2) tendon reactions still absent.

Case III.—E. S., aged fifty-five, formerly a soldier. Present illness began about eighteen years ago, the first symptom being an attack of shooting pains in both legs. This came on after exposure for a whole night on sentry duty to a temperature of −49° C., that is −56° Fahr. Since then the pains used to return in bad weather, but were mitigated after a course of brine baths in 1876. Since January 1880 they have again been very intense. No syphilis. He has four children, having married at the age of twenty, and previous history of excess "in venere" though not "in baccho." When in military service he once fell from a cart on to his back and lost consciousness; the seat of injury was painful for a couple of days after. Patient (1) is unable to walk or stand; (2) has severe shooting pains, as well as a cold, creepy sensation in the legs; (3) is blind; (4) suffers from constipation and incontinence of urine; he has also lost sexual power, sleep, and appetite. He is a man of average build, and looks intelligent and truthful. His habitual attitude is lying on his back, being unable to sit up for more than five minutes at a time or to lie on either side, on account of the pains in his legs. Since August 1881 he can stand only when supported on either side. Bones and joints are normal; no spinal tenderness. Muscles very flabby, the gastrocnemii especially having wasted recently. Patient says that they have decreased. Integuments bloodless and flabby. Pigmented round scars, occasionally adherent to the periosteum, and from one to three cms. in diameter, are scattered over the shins—said to be the remnants of abscesses twenty-five years ago. Nails thick and laminated, and looking somewhat like split nutshells. Motion of arms free and precise. In the lying posture legs raised, or drawn up with effort, the leg movements being jerky, precipitate and excessive. On trying to touch the big toe of one foot with the heel of the other, patient shoots out the leg so wildly as to bring it into collision with bystanders, whilst he fails to reach the desired spot.

The cremasteric and abdominal (tactile) reflexes weak and delayed. Latent time of musculo-cutaneous "pain-reflexes" very protracted.

Right big toe, latent period	1·414″
Left ,, ,, ...	1·916″
Right index finger, latent period	0·147″
Left ,, ,, ...	0·137″

Knee jerk absent; cutaneous vaso-motor excitability slight. Sensibility to pain deficient in the legs, and trunk below level of nipples, becoming less from above downwards so that the soles are insensible even to severe pricks. Great delay in transmission of painful impressions. Tactile sensibility is not so deeply affected. The contact of a hair pencil is least felt at the soles and palms. Thermic sensibility is greatly diminished; no difference in temperatures between 8° and 20° R., nor between 20° R. and 29° R. noticed, but 33° R. and 36° R. are discriminated. The muscular sense and sense of position are entirely lost in the lower extremities. When patient's legs are lying parallel to each other he thinks they are crossed and *vice-versâ*. He cannot tell the position of his leg when it is raised to an angle of 45°. The muscles of the lower extremities react feebly to induced currents; those of the arms do so more readily. Galvanic reactions of muscles and nerves weak though normal in their order of occurrence. Besides pains there are formication and numbness in the legs and alternate feeling of cold and heat in the fingers of both hands. Bowels act only every five or six days; scybala; urine pale, alkaline, sp. gr. 1028, no sugar, but a ¼ per cent. of albumen, and traces of indican, besides pus corpuscles and vesical epithelium. Micturition takes place from fifteen to twenty times daily.

Patient sleeps badly and his appetite is poor. The organs of hearing, smell and taste are intact. Vision began to fail in 1877, at present patient cannot distinguish a window by daylight. Optic discs atrophied. Loss of sexual power since two years.

The lungs are emphysematous. The heart is enlarged in the transverse direction; the sounds at the apex badly defined, impure; over the aortic area a pre-systolic murmur is heard. The radial and temporal arteries are thickened; the latter are also tortuous. The pulse (84) is hard, incompressible. The liver projects below the costal margin but does not seem to be hypertrophied.

Diagnosis.—Tabes dorsalis, emphysema of the lungs, dilatation of the heart, arterio-sclerosis.

In consequence of the changes in the vascular system I did not venture to suspend this patient, but resolved to try the effect of treatment by extension after Volkmann's method. The patient lay on an inclined plane with his feet raised. Cords were fastened to the feet and then passed over pulleys fixed at the foot of the bed. To the ends of the cords weights were attached. After two weeks' treatment the weights were increased to 10lbs.; the patient no longer complained of pains in the legs. This treatment was carried on in my ward from June till December 10th 1882. Pains were absent during the whole of this period. The patient was transferred to the infirmary where the treatment was continued. Slight return of the pains in March 1883. Sleep and muscular power of the legs improved, but the other symptoms remained much as before.

Case 4.—A. F., publican, at Akerman, aged forty-nine, married, seven children. No syphilis nor drink nor traumatism. No illness, with the exception of an acute attack (the nature of which is unknown) in 1854, after which his right hand was frost-bitten and lost all its fingers except the thumb.

Present illness.—In November 1881 patient drove in a snow-storm, the temperature being below the freezing point. On the road he felt hot and had abdominal pains. The fever continued after he reached home, but the pains soon left him. Twelve days afterwards he was feeling better though weak, when he had severe shooting pains in both arms and in both legs. During four days the pains increased, whilst the limbs became weaker, so that on the fifth day he could not stand nor feed himself. The pains although less intense continued until September 1882. During that period patient had not been free from pain for a single hour. Changes in the weather had no influence on the character nor on the intensity of the pains. Motion in the legs disappeared completely at the beginning of March, 1882. According to patient's statement there was no great alteration of sensibility in the upper extremities, but he could not feel when his feet or his legs were touched. During this time sleeplessness from pain, and loss of flesh; bowels and bladder normal; but sexual feelings lost since the outset.

On admission into the Odessa Hospital on the 15th of July 1882 the patient was found to be of good conformation, though thin and weighing only 46·2 kilos. Bones and joints are normal. Flabby, palish and wrinkled about the face. Patient can be comfortable only on his back, and is unable to sit up for more than an hour, and unless propped up with pillows, the legs lie extended with toes dropped, and no trace of voluntary or reflex motion can be detected. Patient usually rests his arms on the abdomen the elbow joints being flexed; he moves them languidly but extensively, and without any co-ordination. The elbow and knee jerks are absent; plantar reflex very weak; cremasteric and abdominal intact. No musculo-cutaneous "pain-reflexes" in the legs where the bulk of the muscles is much atrophied, the extensor femoris being least affected. Gluteal fairly normal; muscles of shoulder and arm in good condition. In the forearm the flexors are more atrophied than the extensors. Left interossii deeply affected; thenar and hypothenar muscles almost non-existent. The hands have a bird's-claw appearance.

Painful pressure points along great nerve trunks, though calf muscles are tender. Palms and on the soles is of a bright red, constantly moist; no perspiration elsewhere.

The muscular sense is normal in the arms, diminished in the legs. The sensation of locality (Weber's compass) is well preserved in all the limbs. Cutaneous sensibility to pain is undisturbed in the arms; in the legs it is diminished from the knees downwards. Tactile and thermic sensibility are everywhere normal. The muscles, in which atrophy is marked, do not re-act to the induced current; the others feebly so. The galvanic current causes nothing but a slight K O C in the less atrophied muscles. No K C C, A C C, or A O C are observed.

Patient complains of tearing and shooting pains in both arms and legs, the pains coming on in paroxysms; of formication and a sensation of weight in the lower and of a constant feeling of little as possible with the ligamentous and muscular connections of the bones. The fifth and sixth nerves could then be seen passing nearly horizontally through the dura mater and forming a slight convexity directed downwards. On suspension the nerve roots became nearly vertical, forming an acute angle with the spinal cord. The body in a horizontal position measured 145cm., and when suspended 151cm. The increase in the length of the vertebral column alone from the second cervical to the fourth lumbar vertebra was 2·25cm. When the body was horizontal, skin, bone and dura mater were [illegible] ked at the same level in several places. On suspens [illegible] rks on the skin and bone were found to be 14mm [illegible] tho [illegible] e dura mater. Although the strain [illegible] ot marked it appeared to me the [illegible]

In taking these measurements I made use of an apparatus resembling Weber's compasses. The subject when standing was placed in such a position that the heels, sacrum, spine and occiput all touched a vertical measuring rod. A movable piece at right angles to the rod and riding upon it was then lowered till it touched the patient's head. The measurements during suspension were taken by means of two pointers, which could be moved along the measured rod and placed in contact with the desired spots on the patient's body.

The figures in the above t[illegible]ow that the vertebral column takes part in the gen[illegible]ation. That this is not due to the [illegible]ning [illegible]urves alone is easily shown by tak[illegible]h a tape applied to with the shoulder straps. The posterior part of the collar should be more elevated than the anterior—the patient's ears remaining free in the triangles formed by the collar and the straps.[1]

It is also necessary to remark that the treatment should always be carried out by a *medical man*, and not entrusted to inexperienced persons. The patient should be raised and lowered slowly and without jerks. Before raising it is well to see that the strain on the head and shoulder straps is equal, as in that case the patient is able to endure the operation for a longer time by transferring his weight alternately from his head to his shoulders. The first suspensions should not last longer than from one to two minutes.[2]

Suspension influences both the respiration and the circulation. The former becomes costal in type and is accelerated. The patient K., who breathed from sixteen to eighteen times per minute while standing, breathed from twenty-two to twenty-four times per minute on being suspended. In other cases a similar acceleration was noticed; on an average it was equal to four per minute. The pneumo-manometer also showed that both respiratory acts were interfered with. In the case of the same patient, while standing the pressure was equal to —30 mm. of mercury for inspiration and to + 80 mm. for expiration; while the patient was suspended the figures were —10 mm. for inspiration and + 60 for expiration.

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[1] Dr. Motschutkovsky has recently modified the suspension apparatus. The collar not unfrequently compresses the trachea and the great blood vessels of the neck, more especially in stout persons; this gives rise to attacks of syncope, which may assume a dangerous character, to vertigo and to cyanosis. In the new arrangement the collar is replaced by a pad and a chin piece, both connected by straps. The pad is made of hard leather and is applied to the occiput. The chin-piece is also made of hard leather, is cup-shaped, and its concavity is well padded and lined with wash leather. The chin of the patient is received into the hollow. The occipital pad and the chin-piece are connected by means of straps, which pass over the rami of the lower jaw and over the mastoid processes. In this manner compression of the blood vessels of the neck is avoided. The chin-piece can be raised or lowered by means of straps in such a way that the head remains in an easy position, which is a point of the utmost importance.—TRANSLATOR.

[2] In order to avoid attacks of vertigo and syncope during suspension, Dr. Motschutkovsky is accustomed to pay close attention to the state of the patient's consciousness. He does this by conversing with him.

It is important that the patient should not be allowed to rotate about a vertical axis: this is apt to happen when the ropes used are new.—TRANSLATOR.

The pulse rate is also accelerated. In the same patient K., it was equal to sixty-four beats per minute while standing. During the first minute of suspension it was 75 per minute, during the third it was 82, during the fourth 84, during the seventh 86, during the ninth 86.

Sphygmographic tracings show that the blood pressure is raised during suspension: the diastolic wave noticeably approaches the apex of the systolic wave. As the shoulder straps interfere with the circulation in the radial arteries these observations were carried out on the femorals.

From these meagre data it is, of course, impossible to come to any conclusion as to the reason why suspension does good; nor is it clear why it should prove most useful in the treatment of tabes. It is certain that during suspension changes take place in the relative positions of the spinal cord, the nerve roots and the vertebral canal, although the nerve roots do not become noticeably tense. It is probable, moreover, that, as the lower extremities become elongated to the extent of more than 3 cm., the peripheral nerves are stretched, as they are in the operation of nerve stretching. Accordingly, suspension might be supposed to act by effecting dynamic changes in the peripheral nerves after their emergence from the vertebral foramina.

This might be admitted, if the treatment were efficacious only in arresting the progress of the disease. But as some of the symptoms are markedly improved, it appears to me improbable that this can be due to changes in the nerves alone. In all slowly progressing degenerations of the central nervous system it appears to me that improvement can only be brought about by improving the nutrition of those parts of the tissue which are as yet only slightly changed; whether this takes place directly or by increasing the activity of the collateral circulation. Of late novel views as to the physiological pathology of tabes have begun to obtain ground. According to these views (Adamkiewicz) the proximate cause of degeneration of the posterior columns is a change which takes place in the blood vessels. Microscopical researches have shown, that the changes in the tissue begin as foci (which afterwards become blended) situated in the neighbourhood of diseased vessels. It is obvious that, if the nutrition of the affected parts can be maintained by increasing the blood-pressure, these parts may retain their activity for a longer or shorter period. We have seen that during suspension the blood pressure is increased; the blood vessels are at the same time stretched. May it not be that these changes increase the activity of the circulation in those vessels of the cord, which are as yet unaffected?

It is remarkable that recent cases of tabes are not benefited to the same extent by suspension as those of old standing. I am of course far from claiming that tabetics can be cured by means of suspension. But on comparing the results of nerve stretching with those obtained by suspension, I must give preference to the latter. Moreover, suspension does not interfere with the patient's occupation, is not painful, and if certain precautions be observed, is not dangerous. It is true that b s of nerve stretching pain has been relieved at once eral instances. But although this is effected more by means of suspension, the analgesic effect of the l ode of treatment is most marked. None of those tr isorders which sometimes follow nerve stretching (p owing to irritation of the grey substance of the sp rd) have been noticed after suspension.

The conditions which I sidered ontra-indicate suspension are as follows:—

1. Heart disease and dis
2. Sclerosis and aneurys
3. Marked emphysema
4. Cavities in the lungs
5. Previous epile
6. Profound

Patients

than

mir

Frequent (daily or twice a day) and prolonged (twenty minutes) suspensions gave rise to the following bad effects:—

One patient complained of sudden loss of power in the lower extremities. Some had pains in the waist and back. Others suffered from vertigo, loss of appetite and drowsiness. A small hæmorrhage into the sclerotic took place in one patient. Retention of uri e was observed in one case. Finally, the patient F. suffered from spasmodic contractions of the lower extremities (which did not, however, last long).

I have not been able to decide whether the ætiology of tabes have an effect in rendering some cases more amenable to treatment by suspension than others.

In order to avoid fallacies I used no other method of treatment at the same t spension.

The sole purpose aper is to register results obtained in the treat e of the most painful and hitherto most hopeless ses. I look forward to far more extended and va igations in order that the general applicability an e value of the method here described be brought in nt and invested with scientific certainty.

Up to the present tin of application of suspension has not been greatly exte . Motschutkovsky's wards, but his former concsions h peatedly confirmed by more recent obser od results have been obtained in cases especially of the sciatic nerve. Amo treated by suspension, been ble improvement. This markal tschutkovsky and my- Dr. Mc histological changes of ng the estigation will be pub- ve been made in ion acts benefi-

nt has

The

cially in cases of impotence of non-tabetic origin. This subject will be treated by Dr. Feldman in a paper which he is at present preparing for the press.—TRANSLATOR.

AUTHOR'S NOTE, JUNE, 1889.

I have been able to obtain accurate information as to the subsequent history of these patients only in four instances:—

1. The patient, P. M., who is described as case No. 1, is at present following his occupation of assistant to a carriage proprietor. He lives at the distance of more than one verst (two-thirds of an English mile) from the place where he works, and walks there every day. During two summers he was in the habit of going on foot daily to bathe in a salt lake at the distance of about five versts from his residence. His gait is still ataxic, and he is subject at times to various paræsthesiæ, but has not suffered from pains in the extremities. Sexual activity is present. There is no patellar reflex and the pupillary reflex is also absent. There has been no relapse of any kind, although the patient has not been suspended since he left the hospital.

2. The patient, A. F., described as case No. 4, considers himself to be completely cured; he is able to work as before. He continued to improve rapidly after he left the hospital, and since that time has not been treated in any way.

3. One of the twelve tabetics who are mentioned in the table, has remained free from lightning pains in the extremities and from incontinence of urine ever since he was treated by suspension. The patellar and pupillary reflexes are both absent.

4. Finally, another tabetic, also included in the table, was under observation for two years after his course of treatment was completed. He then removed to Moscow and has not since been heard of. During that period there was no recurrence of gastric crises, from which he used to suffer at least once or twice every week. His gait also continued to be fairly good. The rapid progressive deterioration of his eyesight, which existed previous to treatment, was arrested. The patell[illegible]ined absent.

With regard to the question whe[illegible] course of suspensions might not be necessary in[illegible]s, I may state that I have not as yet had occasion t[illegible]h a measure.

Clinical Cases.

CASE OF TUMOUR OF THE RIGHT TEMPORO-SPHENOIDAL LOBE BEARING ON THE LOCALISATION OF THE SENSE OF SMELL AND ON THE INTERPRETATION OF A PARTICULAR VARIETY OF EPILEPSY.[1]

BY J. HUGHLINGS JACKSON, M.D., F.R.C.P., LL.D., F.R.S.,
AND CHARLES E. BEEVOR, M.D., F.R.C.P.

EMILY M., a widow, aged 53, was admitted under our care (National Hospital for the Epileptic and Paralysed), November 25th, 1887; she died December 31, 1887. For nearly all of the notes of the case we are indebted to Dr. Hull. There is nothing very noteworthy in her family history, nor, until this illness, in her personal history. She said that she had had a polypus removed from the uterus some years ago. The first thing in her present illness, about thirteen months before admission, was the occurrence of epileptic fits. As these attacks were very odd, independent accounts of them were obtained by Dr. Beevor, Dr. Hull and Dr. Hughlings Jackson; there was no essential variation, if indeed any, in the several accounts.

The first account given is by the patient's sister; some of the facts stated were of course obtained by her from the patient. The patient was a cook. In the paroxysm the first thing was tremor of the hands and arms; she saw a little black woman who was always very actively engaged in cooking; the spectre did not speak. The patient had a very horrible smell ([illegible]-called "subjective sensation" of sm[illegible] she could not describe (*vide infra*). She had a feel[illegible]ere shut up in [illegible] box with a limited quantity of ai[illegible]is was; only he[illegible] way of speaking of a feeling of suffo[illegible]. Hull's note o[illegible] the paroxysm). She would stand w[illegible]fixed and directed forwards for a few moments, and[illegible]hat [illegible] horrible smell!" The patient did not, so h[illegible]ted, lose consciousness, but remembered everything that happened during the attack; she turned of a leaden colour. The patient told us that she passed her urine in the seizures. There was no struggling, and the tongue was not bitten. She never believed the spectre to be a real person. After leaving her kitchen work she had paroxysms with the smell sensation, but no spectre. She had had these paroxysms ever since, sometimes three in a day, sometimes one in two days.

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[1] Re[illegible]cal Society on February 18th, 1889.

The patient herself gave to Dr. Beevor, before her admission, and to Dr. Hughlings Jackson and Dr. Hull afterwards, essentially the same account of the paroxysms with the spectre and smell. The following is re-description in essential features.

Dr. Hull writes that she said that in the paroxysms she used to see a little black woman who was rather agreeable and was always flitting about the kitchen; she always saw the same woman in every paroxysm. She never thought it was anything but a vision, but was much worried about it. When she had a fit, she used to have a very nasty smell—"burning dirty stuff"; the smell rising after the fit made her feel *suffocated*.

The patient's memory had been getting worse for six or seven months, but no other mental symptoms were noted except those which were paroxysmal—those already stated. It is to be particularly noted that she had not complained of headache during her illness—a noteworthy fact in a case of large cerebral tumour; she had pains in the neck for the last week or two. Her illness had been preceded by trouble, loss of money—not a likely thing to have been the cause, or, at any rate, more than the exciting cause, of any such symptoms as she had. A week before admission she was noticed to get gradually weak of the left side, and it was observed that the face was "lop-sided," and the left eye was noticed to close. She complained of a leathery feeling on the left side of the body.

On admission, November 25th.—A very fat woman; she lay mostly in a torpid condition. After a good deal of rousing she could be made to walk and could walk fairly well when urged along, yet without decided paralysis of the limbs there was a tendency to go to the left. The patellar, triceps and wrist jerks were equal on the two sides; there was no ankle clonus; the right plantar reflex was greater than the left, but both were active. There was a little dropping of the left side of the face, especially seen when she spoke. The tongue was protruded slightly to the left; it was not tremulous. There was no ocular paralysis, no nystagmus; the pupils were of equal size and their reactions were normal. There was no hemianopia. Sensation, tested by light touches and by pin pricks, appeared to be normal and equal on the two sides of the body. The optic discs were normal. A few days later (December 1st), it was found that she could read fairly well with spectacles, but did so as if it were a great trouble, and spoke in a slipshod, slovenly kind of way. (There was never any aphasia, nor any real difficulty in articulation; nothing more in her speech than inertness, as in all else.) Taste and smell were normal. To return to the account of November 25th: there was no obvious defect of hearing, she being able to hear on both sides. No trustworthy comparison between the ears could be made on account of her condition. There was no visceral disease discovered, and the necropsy revealed none. It may be well to say that she kept her bed until death, except that she was occasionally got up, as will be mentioned, to test her legs; as will be seen, she had the delusion that she got up to walk at other times.

November 27th.—She disturbed the other patients in the night for some time by calling out for chops. In the morning she did not remember anything about it.

December 1st.—It was to-day that her reading was tested and that hemianopia was excluded. She walked half the length of the ward, but had to be supported; she leant heavily against the table and chairs, and would, unhelped, have fallen; she made no attempt to save herself. She took food well: she said she had no pain anywhere.

December 8th.—She said that this morning she had a fit, and that it consisted in a suffocating feeling; she had also a "nasty dreadful smell," but she could not describe it more.

December 9th.—This morning, at 7 a.m., she said she wanted to get up and go out. She said she was convinced that she was perfectly able to walk, and said that she did walk yesterday. She said that she sometimes got up, and she complained that she was flung back on her bed; she spoke in an aggrieved way. When got up it was found that she did walk a little better than when last tested; she still has a marked tendency to fall to the left, and would fall to that side if not supported. She says, notwithstanding, that she is quite able to walk.

December 19th.—She says that she thinks she went for a walk yesterday. Is very lethargic this morning and can scarcely be got to say anything. No optic neuritis. Urine acid, no albumen.

December 23rd.—The patient has been getting into a still more torpid condition the last few days. The left arm and leg nucleus lenticularis was much compressed and flattened by the growth, and the internal capsule seemed to share in the compression. On making a frontal section of the brain at the level just in front of the pons (Fig. 5), all the central white matter of the right temporo-sphenoidal lobe was seen to be involved. The lesion lay just outside the descending cornu of the right lateral ventricle. The cortex and white matter of the hippocampal and first and second temporo-sphenoidal convolutions were not affected; the cortex of the third nporo-sphenoidal convolution was not involved, but its al white matter was partly invaded by the growth.

ne level of a frontal section through the middle of ons (Fig. 6), the lesion is seen about the size of a enny piece just outside and below the descending co the lateral ventricle and the cornu ammonis; it es the inferior part of the central white matter of mporo-sphenoidal lobe, but it does not extend to the co ions or their central white matter.

nd this point the lesion rapidly diminished in size an hardly visible in the next section, which was made ab lf an inch behind the preceding one.

icroscopic examination by Dr. Colman, the resident officer to the hospital, the growth was found to be ound-celled rcoma.

vill thus be s ve have here a tumour which is ed in one spc the extreme anterior end of temporo-s lobe, the part which is called campal lo which ins the nucleus been destroyed, there would not have been any cells remaining to register the irritation caused by the growth, and to produce in the mind of the patient the sensation of a "horrid smell."

The growth had also pressed upwards upon the right lenticular nucleus and the internal capsule of the same side, and this would account for the weakness of the left side of the tongue and the protrusion of this organ to the left, and also for the drooping of the left side of the face—symptoms which were noticed on the patient's admission. is important to note that although on admission there no absolute paralysis of the left arm and leg, they subs ly became completely paralysed, due, no doubt, to the al growth of the tumour. The fibres in the internal ule which pass down from the cortex for the face and to are more anterior in the horizontal line than those for t bs, and this would perhaps account for the face being ted before the limbs.

Remarks by Dr. Hughlings Jackson.—This case sts me especially as presenting epileptic fits with what I he "dreamy state" (commonly called "intellectual a in association with a crude sensation of smell. It is group of epilepsies on which I remarked in a 'BRAIN,' July, 1888 ("On a Parti Variety of E Intellectual Aura"). The cas ed *to in a fo* on the first page of that article

The case is of great value ce bearing sation in the remarks on the variety of epilepsy, of which this case is one example. I once more draw attention to Dr. James Anderson's report of the case of one of his patients, who had this variety of epilepsy and a crude sensation of taste—'BRAIN,' October, 1888.

Remarks by Dr. Beevor.—Besides the case reported by Dr. James Anderson, to which reference has already been made, Dr. Ferrier has referred us to two cases in which there were olfactory symptoms during life due to intracranial growths. The first case is one by Sander,[1] described as "Epileptische Anfälle mit subjectiven Geruchs Empfindungen bei Zerstorung des linken Tractus olfactorius durch einen Tumor." Here the patient had fits which were preceded by the warning of a "dreadful disagreeable smell," and he then had chewing movements of the jaws and spitting of saliva,[2] and later on he had convulsions about the face but not in the limbs. The patient became blind, and his mental condition very obtuse, so that it was never possible to test his sense of smell on the two sides. On post-mortem examination there was found, in the middle fossa of the skull, a tumour (glioma) the size of half a rge apple, situated on the under surface of the brain, at
of the left frontal and temporal convolutions.
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not stated);

temporo-sphenoidal lobe seemed to have been involved, the growth was so large as to have destroyed the left olfactory tract, so that it would be impossible to say whether the attacks were not caused by pressure of the growth on this tract; it was also impossible to ascertain whether the sense of smell was intact on the two sides.

Sander refers to other cases by Lockemann,[1] Westphal,[2] Schlager,[3] in which olfactory auræ preceded the fits, but in all these cases the olfactory bulb or tract was affected either by tumours growing either in them or from the overlying frontal lobes.

In none of these cases did the lesion occur in the temporo-sphenoidal lobe without involving the olfactory nerves, and they do not afford any direct evidence of the localisation of the olfactory centre in the tip of the temporo-sphenoidal lobe.

The second case referred to by Dr. Ferrier is one by Dr. McLane Hamilton—"On Cortical Sensory Discharging Lesions (sensory epilepsy)."[4] Here a woman, aged forty, had attacks dating from her tenth year, when she had a fall on to her head. In the attacks she frothed at the mouth, became livid and was convulsed for a great time, b[illegible]e the fits she nearly always had a peculia[illegible]nly perceived a disagr[illegible]our, son[illegible]times of a fœtid chara[illegible]sory warnings. Sh given in this paper, the lesion seems to have involved the inferior surface of the temporo-sphenoidal convolution extending to the anterior end, and affecting especially the third temporo-sphenoidal and the uncinate convolutions; but to what extent this latter was involved is not mentioned, nor is it stated whether the disease involved the uncus itself, the cornu ammonis, the hippocampal lobule, or the inner surface of the temporo-sphenoidal lobe.

The last case is exceedingly good for the purposes of localisation, as the lesion, as far as it is described, was very definite, and the olfactory nerves were found to be intact. Unfortunately, however, there is no mention as to the condition of the sense of smell on the two sides during life.

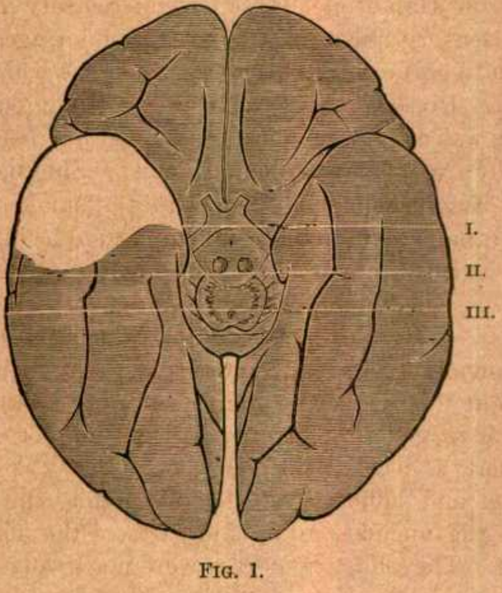

FIG. 1.

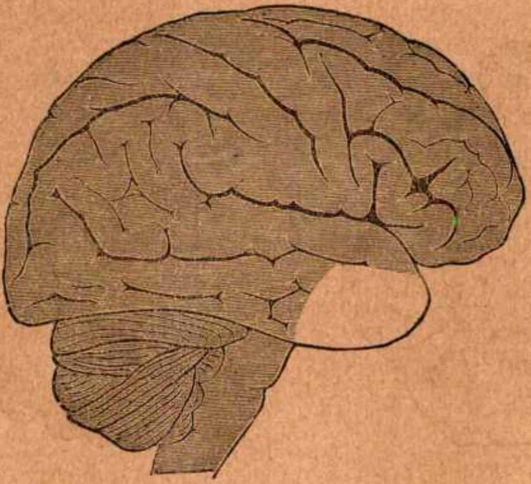

FIG. 2.

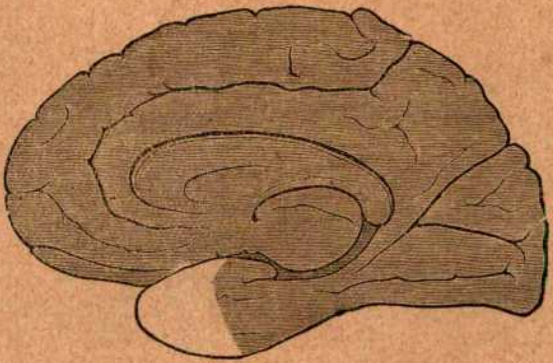

FIG. 3.

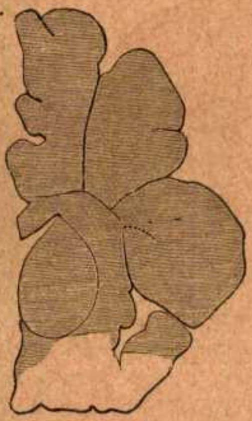

FIG. 4.

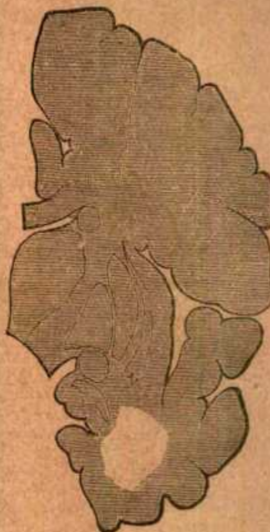

FIG. 5.

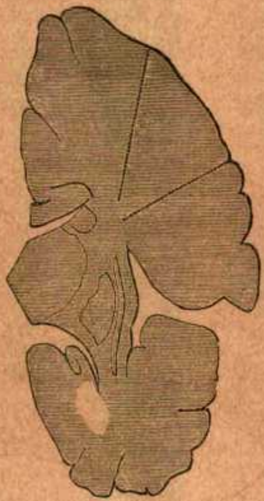

FIG. 6.

EXPLANATION OF THE FIGURES.

The part occupied by the tumour is shown as it appeared on the surface of the brain, and also in frontal sections.

The horizontal transverse lines ruled across Fig. 1 indicates the levels at which the frontal sections, shewn by Figs. 4, 5 and 6, were made.

Critical Digests.

CEREBRAL LOCALISATION IN ITS PRACTICAL RELATIONS.

BY CHARLES K. MILLS, M.D.

(*Continued from p.* 288.)

A CAREFUL, elaborate, clinical study of hemiplegias, monoplegias and aphasias will eventually enable us to separate with considerable certainty lesions of the cortex from those of the centrum ovale, capsules, and ganglia. We will do this by relating the symptoms found not only to lesions of the cortex and the projection system of fibres, as is too commonly the restriction placed on our studies in this direction, but also to lesions of commisural and association fibres. I am convinced that a lack of consideration of these commisural and association fibres is at the bottom of much of our confusion in analysing certain cases. Very few lesions are absolutely cortical. Many of those which are generally regarded as cortical involve to a greater or less extent the sub-cortex. As every convolution of the cerebral surface is connected with some other, and probably with many other convolutions, some association fibres must nearly always be destroyed in these cases.

Differential Diagnosis, particularly of Jacksonian Epilepsy—Dural and other Reflex Epilepsies.

Sufficient diagnostic difficulties are still present to make it important in the light of the tremendous impetus towards operations to carefully examine all questions of differential diagnosis. We should know in connection with cerebral motor localisation, whether certain affections do not simulate cortical epilepsy so closely as to sometimes endanger exact diagnosis. In certain motor and especially spasmodic affections for example we have striking resemblance between affections clearly of reflex origin and those as demonstrably central. Trigeminal epilepsies, whether dural, facial, dental, nasal, pharyngeal, laryngeal, or excitation of the cortex. Latent period, form, and duration of curves. Convulsions provoked in the same way and studied with similar apparatus. March of the epileptic seizure and its characters, whether complete, incomplete or abnormal. Study of the characters of the disorders of the circulation of the pupils and of secretions which occur on stimulating the cortex of a curarized animal, and conclusion that in these cases the seizures are epileptic, the organic system alone participating in the epilepsy. Effects of lesions of the brain upon motor functions. Theoretic considerations; proper excitability of cortex as peripheral impressionable field.

CORTEX : VISION.

24. Case of Cortical and Sub-cortical Disease of the Occipito-angular Region producing Hemianopia. SHARKEY (*Trans. Ophthalmo. Society*, viii., p. 304).

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25. The Relation of the Occipital Lobes of Newborn and Young Animals to Movements of the Eyes. DANILLO (*Vorläufige Mittheilungen Wratsch, No.* 48, 1888).

26. Influence of Cortical lesions on Vision (*concluded*). LANNEGRACE (*Archives de Médecine Expérimentale, &c.*, 188[illegible], ii., p. 289).

Experiments on monkeys. 1. Lesion of one occipital lobe seems to have no effect on vision (agrees with *Ferrier, Yeo, and Munk*). 2. Lesion of one gyrus angularis affects vision of opposite side, but effect is transitory. 3. Lesion of the gyrus angularis and occipital lobe affects vision with both eyes, producing crossed amblyopia as well as homonymous hemiopia (agrees with *Ferrier*, and *Yeo*). 4. Lesion of the Rolandic convolutions disturbs vision. 5. Successive lesion of both angular gyri results in no loss of sight. 6. Destruction of both occipital lobes causes disturbances of vision but not blindness. 7. Destruction of both occipital lobes and one angular gyrus does not cause complete blindness (in accordance with *Ferrier*). 8. Destruction of both occipital lobes and both angular gyri does not necessarily produce complete loss of vision as *Ferrier* and *Yeo* assert. In one case L. obtained this result, in another case not.

L. concludes that to completely destroy vision in monkeys the injury must extend farther, into the motor zone. In chapter four L. gives a theory of hemiopia and amblyopia, and the rest of the paper is occupied with a discussion of the course of optic fibres.

stimulating the occipital cortex. U. considers these fits as distinctly of cortical origin, although distinguished by one-sidedness. He thinks they are due to the propagation to the motor area of stimuli originating in the occipital cortex. These fits are liberated by stimulating the most posterior region in which such a stimulus is effective—the posterior second longitudinal convolution on the inner side of the vision sphere. Stimulation with short weak currents induces movements of the eye balls to the opposite side—later widening of the pupil and opening of lids. With longer action an epileptic fit is liberated. The fit begins with nystagmus and spreads in the order in which the current would extend supposing it to have passed from the occipital region to the frontal cortex in the first place and there to have originated the movements of the eye balls by cortical action in this spot in the front of the motor area : nystagmus, movements of ear, orbicularis, muscles of cheeks, tongue and extremities; bending of vertebral column, widening of pupils, and then extension of the fit to the opposite side of the body. Sometimes movements of posterior extremities precede the movements of orbicularis. If the orbicular centre is severed from the centre for the lateral movements of the eye by a sharp cut the course of the fit is altered, and contraction of the orbicularis ends the one-sided fit. The fit affects one side before it spreads across to the other.

Paper is illustrated with photograph showing alterations of fit produced by cutting away motor area (in dogs). If whole motor area is removed movements of orbicularis and ear of the opposite side follow the nystagmus—then a pause as long as would have been required for the spasm to travel down to the extremities, and after this the spasm passes across to opposite hind foot and gradually ascends body.

22. The Question of the Epileptogenous property of the Posterior Part of the Brain. ROSENBACH (*Neurologisches Centralblatt*, 1889, *No.* 9, pp. 249-257).

Contra Unverricht (cf. supra) Rosenbach concludes that because epilepsy is no longer produced by stimulating the occipital cortex after removal of the motor area, it is due, when this area is intact, to spread of stimulus. U. believes that it has its proper origin in the spot of cortex stimulated. The paper is devoted to arguments against U..

EPILEPSY.

23. Lessons on the Motor Functions of the Brain and on Ce[illegible] FRANCOIS-FRANCK (Paris, 1889).

Graphi[illegible] vements produced by electrical the longitudinal thickenings. Subsequently a continuous ventral commissure, anterior and posterior commissures are formed. The fibres of the optic nerve are intimately connected with the main bundle of fibres in the region of the primitive transverse thickening. The anterior band of nerve-fibres corresponds in situation with *Edinger's* "Commissur der basalen Vorderhirnbundel." Its large size indicates that it was one of primary importance. The commissure in the adult brain is probably a rudiment of the same with changed relations and functions.

COMPARATIVE ANATOMY CORTEX.

16. Convolutions and Sulci of Great Brain of the Dog. ELLENBERGER (*Archiv. f. wiss. u. prakt. Thierheilkunde*, xv., 3 & 4).

CORTEX: PHYSIOLOGY.

17. The Results of Section of the Association Fibres in the Dog's Brain. EXNER AND PANETH (*Pfluger's Archiv.* xliv. p. 544, 1888).

CORTEX: IRRITABILITY.

18. Excitability of the grey substance in the motor area of the cerebral cortex. ADUCCO (*Archiv. ital. de Biologie* xi. 1, p. 122, Jan., 1889.

CORTEX: LOCALISATION.

19. The Physiology of the Motor Area of the Cortex BECHTEREW (*Russian Arch. of Psychiatry* ix. 2, 3, and x. 1, 2, 1889.)

CORTEX: MOTOR.

20. The Nervous Centres in a Man from whom a limb had been amputated. BIGNAMI AND GUARNIERI (*Boll Della R. Accad. Medic. di Roma. Abs. in Archivio ital. per le Malat. Nervosi* xxvi. p. 40, 1889).

A man 45 years old, from whom left arm had been amputated eleven years before. Atrophy of posterior column and whole of grey matter in cervical region on the left side and of the right column *of Gower*. Atrophy of the cortex of both central convolutions on the right side.

CORTEX: EPILEPSY.

21. The Relation of the posterior part of the Cortex to the Epileptic Fit. UNVERRICHT (*Deutsch. Arch. f. Klin. Med. Bd.* 44, 1889).

There has been some controversy between U. and Rosenbach as to the meaning of the convulsions which may be provoked by ment of its sensory nuclei and tracts, with the sensory elements of the vagus and trigeminal systems. The fine fibres of the urodele cerebellum are in part decussating tracts of the auditory nerve, and the coarse fibres are non-decussating descending tracts of the trigeminal nerve. The cerebral commissures contain decussational fibres. There is a very close similarity between the amphibian (urodele) and dipnoan brain, both in external and internal structure.

COMPARATIVE ANATOMY: FISHES.

10. The Homologies of the Inferior Lobes of the Brain in Fishes. CHATIN (*Comptes Rendus*, cviii., 12, p. 628).

COMPARATIVE ANATOMY.

11. The Nervous System of the Decapodous Crustaceans and its Relation with the Circulatory Apparatus. BOUVIER (*Annales des Sci. Nat.* vii., 1 and 2, pp. 72-96, pl. 7, 1889).

COMPARATIVE ANATOMY.

12. Neurology of the Prosobranchiata. BROCK (*Zeitschr. f. w. Zoologie*, xviii., 1, pp. 67-84, pls. vi. and vii., 1889).

COMPARATIVE ANATOMY.

13. Ventral Nervous Mass of Fissurella. BOUTAN (*Archives de Zool. Exper.* vi. 3, pp. 375-433, pls. xxi., xxii., xxiii., 1888).

COMPARATIVE ANATOMY.

14. The Nervous System of Amphiptyches Urna. MONTICELLI (*Zool. Anzeiger*, 302, p. 142, 1889).

DEVELOPMENT.

15. Development of Amphibians, chiefly concerning the Central Nervous System, with additional Observations on the Hypophysis, Mouth, and the appendages and Skeleton of the Head. ORR (*Quart. Jour. Micros. Sci.* cxv., pp. 295-325, pls. xxvii.-xxix., *Dec.*, 1888).

Embryos of Amblyostoma, Triton, and Rana. Conclusions:—The central nervous system of amphibians first appears as a transverse epiblastic thickening dorsal to the mouth-fusion. This transverse thickening is continuous with elongated thickenings, which lie on either side the mid-dorsal line. The transverse thickening is the cause of the primary cranial flexure. It corresponds to the region at the base of the brain between the infundibulum and optic groove. The first nerve-fibres are formed on the inner side of

9.—Internal Structure of the Amphibian Brain. OSBORN (*Journal of Morphology*, vol. ii., No. 1, pp. 51-96, pls. iv.-vi. 1888).

Results of observations on Amphiuma, Cryptobranchus, Necturus, Siredon, Proteus, Rana and Siren. The following are some of the most important results arrived at:—1. The determination of the chief motor and sensory nuclei of the fifth and tenth pairs, which enables him in some degree to homologize the intra-axial elements of the vagus and trigeminal systems, and demonstrates the independence of the auditory system and system of the motor nerves of the eye-ball. 2. The discovery of a new tract and nuclei—the *fasciculus communis*—common to the tenth, ninth, and seventh (or eighth) pairs. 3. The determination of the relations of the posterior longitudinal fasciculus (uncrossed Müllerian fibres) to the eighth, sixth, fourth and third nerve tracts and of the nucleus of the latter nerve with the fibres of the posterior commissure. 4. The passage of a portion of the descending trigeminal tract through the cerebellum, and the direct connection of this bundle with the large mesencephalic nucleus. 5. In the encephalon the determination of the direct motor tract to the prosencephalon and of direct sensory tracts to the mesen- and dien- cephalon.

The brain forms are described in great detail and well illustrated. It is impossible to give an abstract of the anatomical work, but certain conclusions may be mentioned. Osborn suggests that the cerebellum is primitively intersegmental, and secondarily acquires a functional importance equivalent to that of the other segments. The early constriction of the brain roof, which gives rise to the four vesicles, is for the accommodation of three nerve-fibre tracts decussating dorsally, viz., the superior and posterior commissures and the cerebellum, which in their primitive condition have a serial homology. With regard to the intra-axial origin of cranial nerves in amphibia, Osborn comes to the following "provisional conclusions:" (1) That there is a close similarity between the disposition of the nuclei and tracts of the V.-VII. and IX.-X. groups—the nerves of these groups being complementary to each other, and together apparently containing fibres from two sensory nuclei and from two motor nuclei. The extreme dorsal and ventral nuclei are composed respectively of unmistakeable sensory and motor cells. (2) III., IV., and VI. pairs form a special system not homologous with (1). (3) VIII., as a nerve of special sense, has either no homology or an incomplete homology in the arrange-

WEIGHT.

6. Brain Weight of New-born Children. MIES (*Wiener klin. Wochenschr*, 1889, 2, p. 39).

EVOLUTION.

7. On the Relation between the Structure, Function, Distribution and Origin of the Cranial Nerves; together with a Theory of the Origin of the Nervous System of Vertebrata. GASKELL (*Journal of Physiology*, x. 3, pp. 153-211, pls. xvi.-xx, *April*, 1889).

Cf. Paper in this Journal xlv. and xlvi., July, 1889, in which Gaskell explains his views as to the origin of the vertebrate nervous system. In this paper Gaskell also gives an account of the histological character of the third and fourth nerves, showing that they contain amongst their fibres tissue not found in other nerves. This tissue appears vestigial, representing apparently degenerated "stationary" or root ganglia. Nerves III. and IV. are therefore complete segmental nerves, the sensory parts of which have degenerated and been replaced in their distribution by sensory elements of V. The ramus ophthalmicus profundus is not a separate dorsal root, but a part of V. which has come up from a hinder segment for the purpose of replacing lost sensory nerve roots. III. and IV. are the primitive segmental nerves of the first and second segments.

VII. also shows traces of a lost sensory ganglion. It therefore corresponds to a complete segmental nerve. The fibres from the back of the oculo-motor nucleus which pass down the posterior longitudinal bundle and out of the brain, with the roots of VII. to supply the m. orbicularis palpebrarum, constitute the splanchnic ganglionated efferent portion of the oculo-motor and trochlearis segments. In his earlier papers Gaskell regarded the large fibres of VII., which form a group by themselves (somatic efferent division of the nerve) as destined for mm. stapedius et levator palati. This he finds is an error. He is unable therefore to find the somatic efferent fibres of the hyoid segment.

The paper is illustrated with numerous reproductions of sections, drawings and diagrams in colour.

COMPARATIVE ANATOMY.

8. The Brain of the Chimpanzee. GIACOMINI (*Atti della R. Academia delle Scienze di Torino*, xxiv. 24 pp. 4 *lithographs, June* 23, 1889).

Detailed account of surface of corpus callosum and base of brain.

morbid states characterised by a considerable weakness of the patient, such as hæmorrhages, physical or intellectual over-exertion, onanism or venereal excess, aneury and chlorosis; intoxications (particularly that by the chloroform in surgical anæsthesia) or chronicals (lead, mercury, sulphuret of carbon) or acute diseases of the genital apparatus; several diseases of the nervous system (sclerosis in disseminated patches, tabes dorsalis, Friedreich's disease, progressive pruritive myopathy, and low compression of the marrow by Lott's disease). This list, already long, is as the author shows, far from being at an end, for the simple reason that the exciting causes are but occasional, insignificant, and not at all specific.

It is partly to the demonstrations of this proposition that the second part of the work of M. Georges Guinon is devoted. He establishes first of all that the disease of which he has given so many examples in the first part is in reality hysteria. But he remarks also, in answering criticism made abroad, and particularly in Germany, that if the exciting causes develop hysteria, they can also give life to other nervous disorders, neurasthenia particularly, whose combination with hysteria constitutes some really complex cases that have been and are still falsely interpreted and considered as special cases in the nosography, under the names of traumatic, taxic neurosis, &c. This combination of different affections under the influence of the same causes is easily understood if one takes in consideration that the existing agents, as the author shows us, are but common occasional causes whose influence can be found frequently in general pathology. But it must be noticed that exciting causes can sometimes give a certain impression to the accidents belonging to the neurose. An hysterical man acted on by lead, for an example, will have an hysterical monoplegia presenting at first sight all the characteristics of the real paralysis by lead, but having also those of hysteria, upon which in reality it directly depends.

M. Georges Guinon then studies the different modes of development of the neurose under the influence of exciting causes, and shews us, with examples, that it may appear several years after their action. Finally he tries to define the mode of action of the exciting causes. In certain cases the outbreaks are caused by phenomena of *auto-suggestion* occurring to the patient against his will. In other cases that are not to be justified by this explanation, the author admits the hypothesis of something

Reviews and Notices of Books.

Les Agents provocateurs de l'Hystérie, By GEORGES GUINON, *Thèse, Paris*, 1889. 1 vol. in 8, 392 pages. Librarie du Progrès Médical, and Lecrosnier et Babé, editeurs, Paris.

THIS work, done under the direction of Professor Charcot, is consecrated to the study of the causes that preside over the growth of hysteria, and also to the different modes of development of neurosis under their influence.

The first part, exclusively clinical, contains the enumeration of the different *exciting causes* ("agents provocateur"), with numerous observations in their support. As to what concerns each of them the author has attempted often with success to find in the old authors some examples of hysteria developed under the influence of exciting causes, the action of which was unknown at the time of the outbreak. In other cases on the contrary hysteria was denied and the nervous disturbances that characterise it were regarded as proceeding from some exciting disease of which they constituted, according to the authors, only a symptom. M. Georges Guinon shows us that hysteria, produced by a certain number of different causes, has always existed, and that taken altogether it is through mistakes in doctrine or in interpretation that it has never been judged at its right value.

Let us only enumerate here the different exciting causes that the author has studied, the lack of space preventing us from following him in the particular study he makes of each of them. Amongst them some have been known for a long time; others—and it is a very interesting point—have had their action revealed quite recently through the researches of Professor Charcot. These exciting causes are: *moral* emotions, attempts at hypnotism, nervous shock, earthquake, traumatism, shock of lightning; general and infectious diseases, such as typhus fever, pneumonia, scarlet fever, rhuematic fever, diabetes, paludal infection, syphilis;

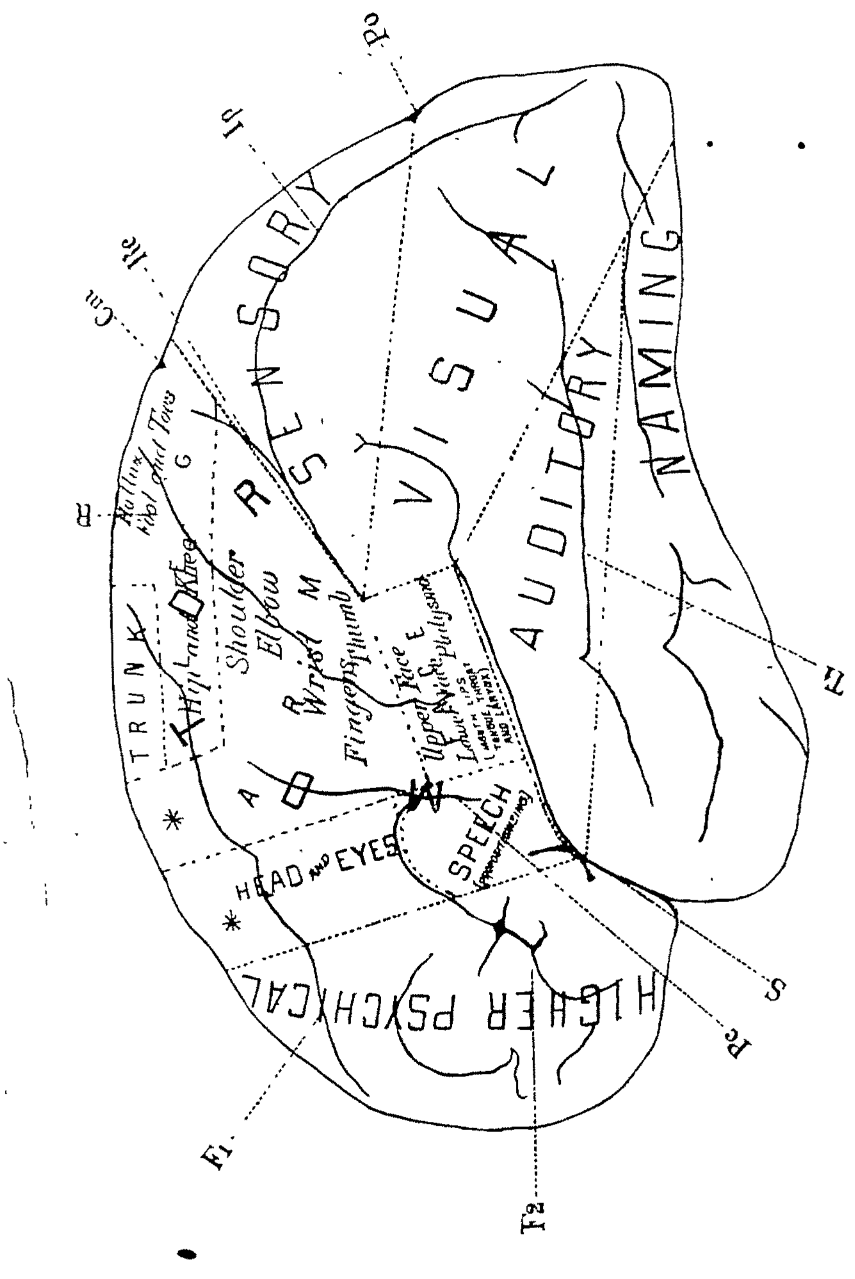

Fig. 6.—Areas of the Lateral Aspect of the Cerebrum, and Sub-divisions of the Motor Area.

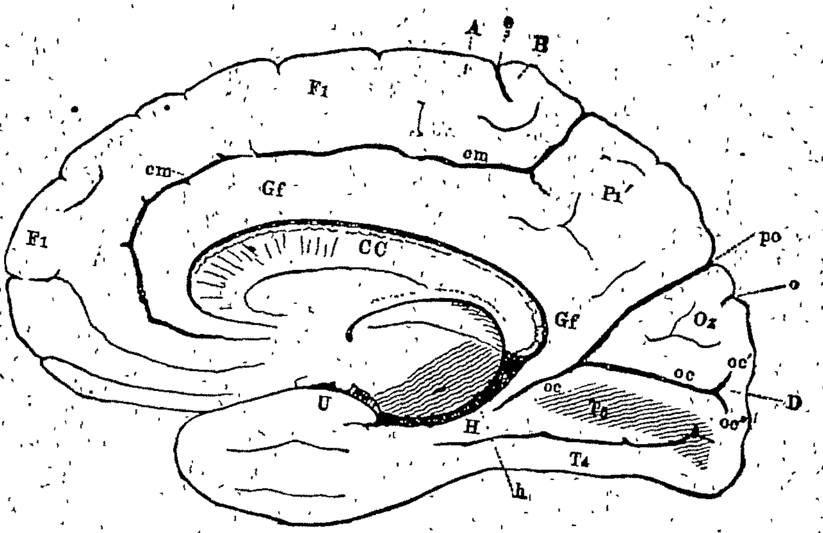

Fig. 5.—Median Surface of Human Brain (Ferrier).

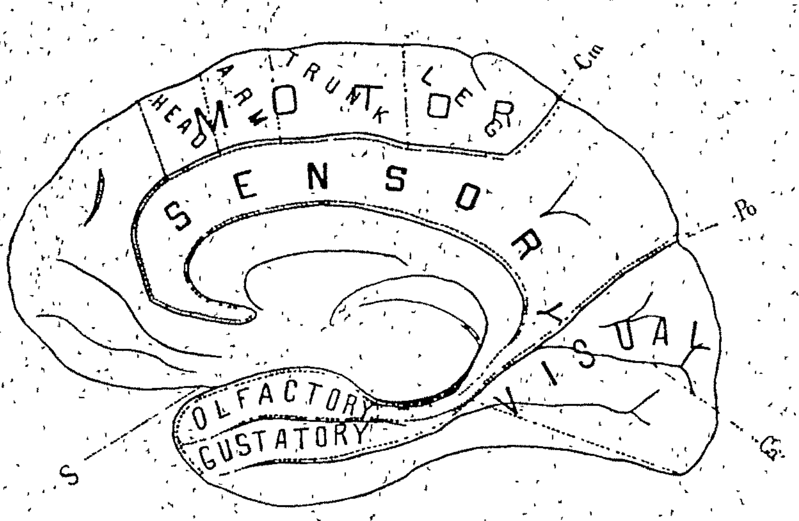

Fig. 7.—Areas of the Mesial Aspect of the Cerebrum.

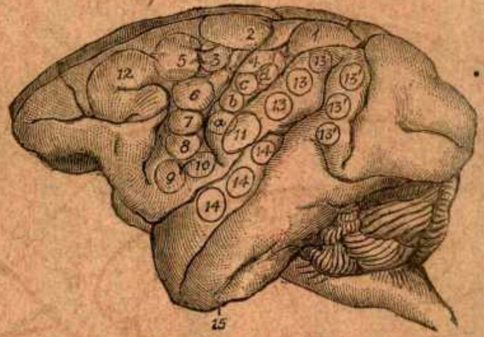

Fig. 3.—Lateral Surface of Brain of Monkey (Ferrier).

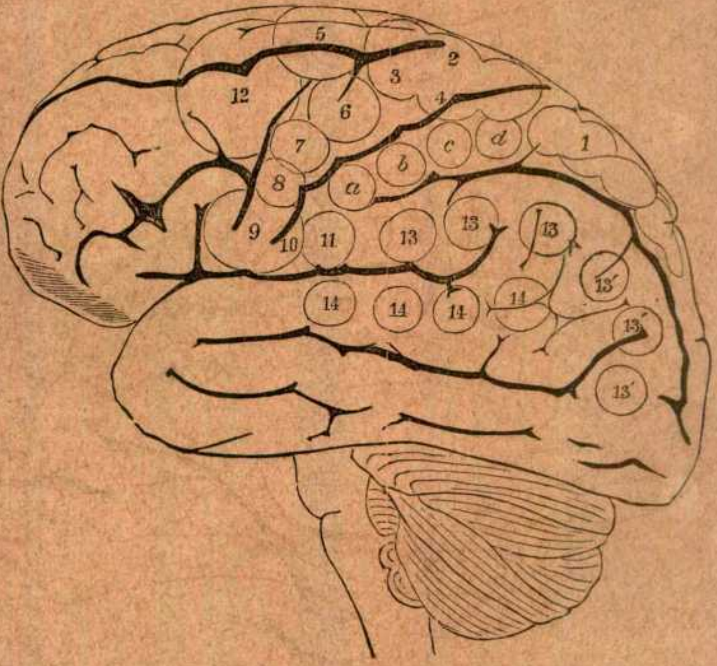

Fig. 4.—Lateral Surface of Human Brain (Ferrier).

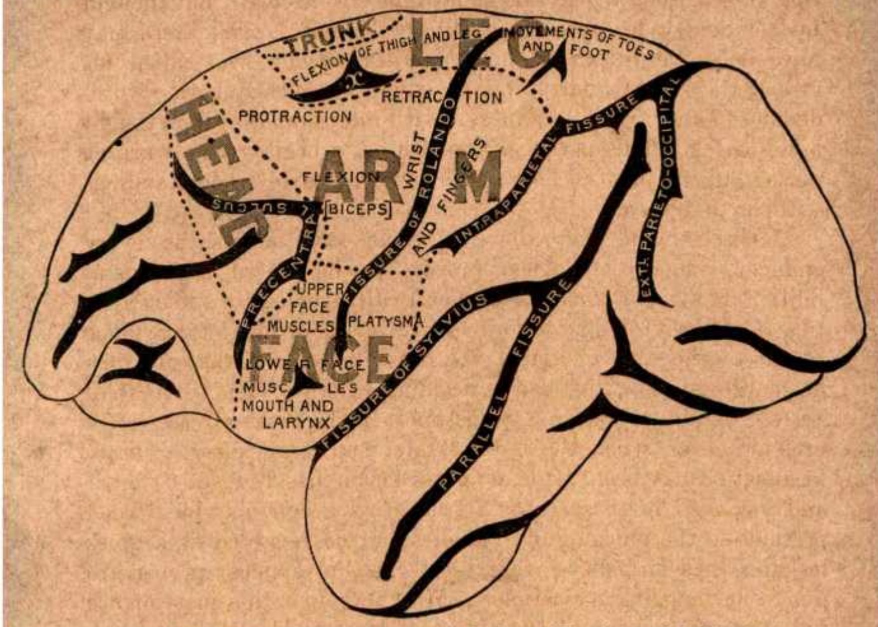

Fig. 1.—Lateral Surface of Brain of Monkey (Horsley and Schäfer).

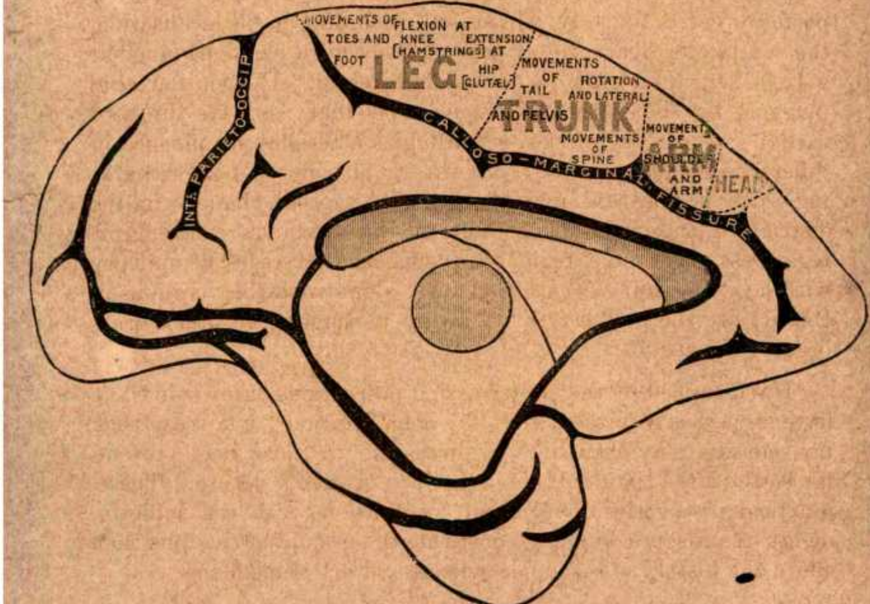
MOVEMENTS OF TOES AND FOOT
FLEXION AT KNEE [HAMSTRINGS]
EXTENSION AT HIP [GLUTÆI]
LEG
MOVEMENTS OF TAIL AND PELVIS
ROTATION AND LATERAL
TRUNK
MOVEMENTS OF SPINE
MOVEMENT OF SHOULDER AND ARM
ARM
HEAD
INTL PARIETO-OCCIP
CALLOSO-MARGINAL FISSURE

Fig. 2.—Median Surface of Brain of Monkey (Horsley and Schäfer).

that this patient was at the same time 'word-deaf,' but thought his temporary deafness was ordinary deafness. Still there may have been word-deafness. In another patient who called his attacks 'losses of understanding,' there was clearly both 'word-deafness' and 'word-blindness,' with retention of ordinary sight and hearing. This patient's attack used to begin with a warning noise, but he has recently had his 'losses of understanding' without that warning."

Jackson holds that there is some local disease in every epilepsy, some pathological process productive of high instability. His views on the arterio-cortical pathology of some varieties of epilepsy or epileptiform seizures are of great importance to those who are concerned not only in locating the site of a discharging lesion, but also in deciding whether such lesion shall be removed by operation. Sometimes, in the cases already operated on, even when the most careful and doubtlessly accurate localisation has been made no gross lesion has been discovered, and yet even in these cases a true gross lesion may have been present—if the plugging of arterioles can be regarded as a gross lesion. Jackson believes that most cases of epilepsy proper are due to the plugging of arterioles. His views upon this question are full of suggestive value. "Centres of taste and smell," he says, "lie, according to Ferrier's localisation, in the region of the posterior cerebral artery, whilst, still according to his localisation, the centres for hearing and part of the centre for sight (angular gyrus) lie in the region of the middle cerebral. Hence, if arterial plugging be the pathology, it may be that we have different varieties of epilepsy proper, according as arterioles are plugged in different vascular regions. The variety of epilepsy I am remarking upon in the text may be owing to morbid changes in the district of the posterior cerebral. But tumours would grow regardless of vascular regions. I suggest that cases of epilepsy with mixed warning (of smell or taste along with the warnings of colour) are more likely to be owing to tumour or other gross organic disease."

Handicapped by the embarrassing proportions of my subject, I have imperfectly presented it for consideration; but trust that my remarks may open lines of discussion to those here present far better fitted than I to enter the lists in such a debate. These practical discoveries in cerebral localisation, with the achievements of antiseptic surgery, constitute the grandest triumphs that adorn the history of our noble science and art of medicine.

localisation, I cannot forbear to recall here the advice of Hughlings Jackson[1] on the great practical importance of the close study of epileptic seizures. These remarks are made in connection with the discussion of the different varieties of aura—crude warnings of smell and taste, intellectual aura of the dreamy state, temporary word-blindness or word-deafness, noises, flashes of light, hallucinations, &c. No better neurological work, Jackson holds, can be done than the precise investigations of epileptic paroxysms. The efforts should be to describe *all* that occurs in the paroxysms. Epilepsies are as numerous as are paroxysms beginning with different warnings. The warning is the first event from or during the onset of the epileptic discharge; it is the clue to the seat of the discharging lesion. Using the term suggested by Seguin, I might interpolate here that it is the sensory "signal symptom." The discharging lesions have as many different seats as there are different warnings of the paroxysms.

"Before we can make good generalisations," says Jackson, "we must carefully analyse. To group together as 'visual warnings' colour projections, apparent alterations in the distance of external objects and 'dreamy states' with definite scenes, is generalising without previous analysis, and is an attempt to organise confusion; they are exceedingly different things. He who is faithfully analysing many different cases of epilepsy is doing far more than studying epilepsy. The highest centres ('organ of mind'), those concerned in such fits, represent all, literally all parts of the body sensorily and motorily, in most complex ways, in most intricate complications, &c. A careful study of many varieties of epileptic fits is one way of analysing this kind of representation by the 'organ of mind.' Again, it is not, I think, an extravagant supposition that there are, after slight epileptic fits of different kinds, many temporary morbid affections resembling those persistent ones produced by destructive lesions of different parts of the cortex. To illustrate for a moment by epileptiform seizures; there is temporary aphasia after some fits beginning in the face or hand (more 'elaborate' utterances, I think, when the exact starting point is in the ulnar fingers); this is the analogue of aphasia from a destructive lesion (softening, &c.). To return to epilepsy. There is, I am convinced, in or after certain paroxysms of epilepsy temporary 'word-blindness;' certainly in one patient of mine who had a 'warning' by noise. I could not make out from lesion of the fifth nerve or of the centre for cutaneous sensation in the cerebral hemisphere. Close testing in such cases would probably show that the olfactory sense was not absolutely abolished.

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[1] *Brain*, July, 1888.

Various cases have been reported with autopsies in which the presence of an olfactory aura has been accompanied by a lesion of the temporal lobe, and these cases, on the whole, point to the lower convolutions of this lobe as the probable seat of the centre of smell.

Hughlings Jackson has written various papers showing the great importance of studying all forms of aura in order to be able better to localise the lesions in the encephalon. As early as 1876, and again in 1879, he considered the varieties of epilepsy in which not only crude sensations or warnings of smell, taste, &c., were present, but also a more elaborate mental condition which he speaks of as the "dreamy state." As theoretical as such discussions may seem at first sight, he clearly shows that they are of practical value not only in leading to an early recognition of epilepsy, but also in localising lesions in either epileptic or epileptiform cases.

I have had several epileptic patients in whom the attacks were initiated by an odour, usually offensive. Jackson[1] gives interesting histories of three such cases in which attacks were ushered in by a crude sensation warning of smell, accompanied sometimes by other warnings, as epigastric sensations and the dreamy state. These cases are of great clinical interest but are not accompanied by autopsies. He refers, however, to the necropsy of a woman who had had paroxysms with the dreamy state and crude arnings of smell. She had left hemiplegia and doub citis. The autopsy showed a tumour in the right ten idal lobe.

Allan McLa has reported the following case of

Zuckerkandl, in this view, is simply following Broca, who divided all animals into osmatics and anosmatics, or good smellers and bad smellers, and believed that the whole of the falciform or limbic lobe was the cerebral organ of smell. I cannot here go into the anatomical and physiological arguments bearing upon this subject. To one familiar with brain anatomy a knowledge of the relatively large size of the gyrus fornicatus and hippocampal region in an animal like man, in whom the sense of smell plays so comparatively an unimportant part, is an argument of some weight against the views of Broca. In osmatics however the hippocampal lobule or region of the amygdala—the uncinate convolution so-called—is very large, while in the anosmatics it is comparatively small. Ferrier's view is therefore probably correct that the only relationship which undoubtedly exists is between the olfactory bulb and the anterior portion of the hippocampal convolution. The anterior commissure has an anterior and posterior division, which connect respectively the olfactory bulb and the region of the hippocampal lobule and nucleus amygdala, a fact which also tends to prove that the anterior portion of the hippocampal convolution is the cortical organ of smell.

Electrical irritation of the hippocampal lobule or uncinate gyrus in monkeys, cats, dogs, and rabbits furnished Ferrier with significant indications of subjective olfactory sensations. This reaction, the same in all, as described by Ferrier, was a peculiar torsion of the lip and nostril on the same side. The experiment in which Ferrier produced destructive lesions of the hippocampal lobule, were, on the whole, unsatisfactory, but to a certain extent supported the view that this region c centre for smell.

Few autopsies have been recorded oss or diminution of smell has been present as the resul l lesion. Ferrier, however, refers to cases reported , Fletcher and

Tumour of the Facial and Auditory Nerves.

At the meeting of the Neurological Society, of London, March 15, 1888,[1] Dr. Sharkey read notes and showed the brain of a case of tumour of the auditory nerve. During the discussion of this specimen, Mr. Horsley stated that the tumour might have been removed by an operation he had already advocated—namely, incision of the tentorium and ligature of the lateral sinus. I have made two dissections with a view of determining whether a tumour in the intra-cranial course of the facial and auditory nerves can be removed, and have concluded that it could, and best, not by operating above the tentorium as I understand Mr. Horsley's suggestion, but by operating below the tentorium, and then pushing aside or excising an outer segment of the cerebellar hemisphere.

Olfactory Localisation.

The location of the cortical centre of smell is still uncertain, but the pointings are all towards the region of the uncinate convolution and its immediate vicinity. Zuckerkandl, of Gratz, in 1887, published a monograph on the olfactory centre, the work being anatomical and physiological, not clinical. The anatomical portion is largely comparative, considering the brains of twelve varieties of animals besides man. One chapter describes the cornu ammonis region, another the olfactory lobes of animals and man, &c. The writings of Burdach, Treverinus, Huguenin and Ferrier are largely quoted. Zuckerkandl believes, but I think without reason, that the entire limbic lobe is the seat of olfactory sensation. Among other cases, he has collected two in which infants born without the olfactory lobe on one or both sides, showed arrested development of the horn of Ammon, and in both of which the gyrus fornicatus and hippocampus were small.

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occipital lobe was discovered and removed. After death a large fibro-glioma was found occupying almost the entire second and third temporal convolutions, and also invading the central portion of the first temporal, the superior aspect of which was somewhat flattened by pressure. The fourth and fifth temporal convolutions were apparently not greatly encroached upon, the uncinate convolution escaping entirely. The patient had no loss of smell. The occipital cyst was evidently a radiation or a secondary result of the growth, due to softening from obliteration of blood vessels. This case emphasises the great importance of carefully studying pressure and invasion symptoms with reference to such comparatively latent regions as the temporal (particularly the right temporal) and pre-frontal lobes. The pressure in this case was evidently not exercised in the same direction as in the two cases referred to above. In a lesion of a so-called latent region, the pressure and invasion symptoms will depend both upon the nature of the growth, and on the particular direction in which it happens to develop.

[1] *Lancet*, London, April 7, 1888.

Large Lesion of the Temporal Lobe.

With a large lesion of one temporal lobe, as tumour, hæmorrhage or abscess, the diagnosis is best made by a careful consideration of pressure and invasion symptoms in addition to those which are strictly localising in character. A single case recently published by Dr. Bodamer and myself will perhaps best illustrate this mode of diagnosis. The chief symptoms were severe headache, more localised in the temporo-frontal region; pain on localised percussion; impairment of sight and hearing; choked discs; dilatation of the right pupil, and three days before death paralysis of the left arm and paresis of the left leg, and aphasia. The autopsy showed a large lesion centred in the middle of the right second temporal gyre; it was a vascular glioma, having beneath and partly around it a cavity containing detritus and a large and evidently recent clot. It occupied a large portion of the interior of the right temporal lobe, but was strictly limited to it.

Macewen reports an operation on a somewhat similar case, of a lesion definitely localised in the temporo-sphenoidal lobe. A patient exhibiting symptoms of cerebral abscess had on the left side ptosis, stable mydriasis, paresis of all the ocular muscles, with the exception of the external rectus, without external squint; on the right side, paralysis of the facial muscles, which retained emotional expressions to a slight degree, and power to close the right eyelid by an effort of will, although it remained partially opened during sleep. He had also paresis of the right arm, which during the few hours he was under observation before operation, had amounted to distinct paralysis. The leg remained normal, and there was no diminution of cutaneous sensibility.[1]

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with half a dozen other specimens, normal and abnormal, and was examined by several brain anatomists and morphologists, who agreed with me as to the striking appearance of lack of development of the first left temporal. Other peculiarities of the temporal, third frontal, and central convolutions, the island of Reil, &c., were noted, and will be given in the description of the specimen.

[1] While this paper is passing through the press, through the courtesy of Dr. H. C. Wood, of Philadelphia, I have had the opportunity of seeing a case of large lesion of the right temporal lobe which shows how active symptoms of such lesions may be absent for a long period, or if present how they may differ from the pressure and invasion symptoms in the two cases above given. Dr. Wood will publish a full account of this interesting and important case. The patient had had for many months general symptoms of brain tumour, such as headache, choked discs, vertigo, &c., but no paralysis and no loss of hearing that could be detected by ordinary tests. During the absence of the patient from the city, left homonymous hemianopsia developed, with pronounced contraction of the other visual fields. Wernicke's hemiopic pupillary inaction was carefully tested for, but was certainly not present. Operation was performed by Dr. D. Hayes Agnew for Dr. Wood, and a cyst in the right

lesions of the cerebellar hemispheres are present, this results from pressure or encroachment. Bulbar symptoms and symptoms of pressure on the bulbo-spinal tracts, both sensory and motor, are somewhat frequently present in cerebellar lesions, particularly late in the history of a lesion of large size. With pre-frontal lesions of large size, the pressure and invasion symptoms are more likely to be disturbances of speech or smell, facial, brachial or aural paresis, and unilateral spasm. Carefully recorded cases show that nystagmus and conjugate deviation may be present in both pre-frontal and cerebellar cases; but in the pre-frontal cases the latter is more likely to be towards the side of the lesion presumably destructive rather than irritative; in cerebellar cases more probably the reverse. Headache is more likely to be frontal in the pre-frontal, and occipital in the cerebellar. Elevation of local surface temperature and tenderness on percussion have some but no great value in differentiation.

Auditory Localisation.

While auditory localisation remains in a somewhat uncertain state, on the whole, the evidence is in favour of the localisation of the cerebral centres or area, of hearing in the temporal lobe, and probably in its upper portion, that is, in the first or second, or in both the first and second, temporal gyres. The most recent and interesting discussion of this subject has arisen from the reports of the results obtained by Schäfer in his experiments on special sense localisation in the cerebral cortex of the monkey. Dr. Ferrier,[1] in reviewing the paper of Prof. Schafer, attacks the results obtained and the conclusions drawn by the latter, and contends stoutly for the localisation of the centre of hearing in the temporal lobe, and in accordance with his original view, more especially in the superior convolution of that lobe. Dr. Starr, in his consideration of aphasia, will doubtless discuss at length the question of auditory centres, naming centres, speech association tracts and speech centres, and I will therefore only briefly and for the purpose of completeness refer to this portion of the subject. Schafer claims to have more or less completely destroyed the superior temporal gyre on both sides in six monkeys, and in one of the animals to have removed every trace of the convolution on both sides. In these cases he reports that hearing was not permanently affected, so far as it is possible to determine in monkeys, and concludes that the auditory faculty is not localised existence of pre-frontal lesions, because psychical phenomena cannot be studied with accuracy in animals below man. Ferrier[1] however found after removal of the pre-frontal lobe a decided alteration in the behaviour of animals, difficult precisely to describe. They had apparently lost the faculty of intelligent observation Horsley and Schäfer,[2] Hitzig,[3] and Goltz[4] have also observed apparent mental changes. Phenomena such as these do not measure in usefulness for the average diagnostician with such positive objective manifestations as hemianopsia or Jacksonian spasm, but they should be valuable aids in the hands of close observers. Among pressure and invasion symptoms motor aphasia, nystagmus, contra-lateral paresis and unilateral convulsions often occur late, particularly in cases of tumour and abscess. Given these symptoms, if now lesions of motor and visual regions can be excluded, it remains only to differentiate the lesions of the cerebellum and possibly some lesions of the temporal lobe.

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[1] *Brain*, January, 1888.

In cerebellar cases, as shown by the studies of Nothnagel,[5] Seguin,[6] and others, the most positive symptoms are usually choked discs, optic neuritis or secondary atrophy, occipital headache, sometimes increased by percussion or pressure on the occiput or neck, vomiting, often apparently causeless in character; typical cerebellar titubation or other disorders of motion or gait; nystagmus and conjugate deviation; and among pressure symptoms one-sided paresis, occasional anæsthesia, sometimes disturbance of temperature, pulse, respiration, and deglutition.

Some lesions of the cerebellar hemispheres as in cases reported by Hun[7] give no localising symptoms.

It might be of value to attempt a contrast of the usual symptoms produced by lesions in these localities. In pre-frontal disease psychic symptoms of the peculiar character just described are usually present; in cerebellar affections they are as commonly absent, although irritative lesions of any part of the encephalon may, of course, cause emotional mental disturbance. The station or gait is not affected in pre-frontal cases: in cerebellar lesions, either typical titubation or some form of ataxia or staggering is usually present. Cerebellar titubation is characteristic of a destructive lesion of the middle lobe of the cerebellum, and when

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[1] 'Functions of the Brain.' [2] *Op. Cit.* [3] Quoted by Ferrier.
[4] Quoted by Ferrier.
[5] *Topische Diagnostik der Gehirnkrankheiten*, p. 78. Berlin, 1879.
[6] *Jour. Nerv. & Ment. Dis.*, vol. xiv. No. 4, April, 1887, 217-235.
[7] *Albany Medical Annals*, May, 1888.

The conclusion is warranted that there is a region for general sensation, including touch, pain, temperature, and possibly pressure, and location of a limb, which can be divided into special sub-areas for the various distinct portions of the body, and that these regions lie along side of and have close anatomical and morphological relations with corresponding motor areas, but that they are not identical with them. From an anatomical and morphological point of view, and from the facts of physiology and pathology, no part of the brain is more likely to contain these differentiated areas for sensation than the gyrus fornicatus, the hippocampal gyre, the precuneus, and the postero-parietal convolutions.

The Pre-frontal Lobe and the Cerebellum.

For the pre-frontal lobe and the cerebellum our diagnostic guides are gradually becoming more definite. We may not be able with the same absolute confidence from positive symptoms and signs to indicate lesions in these localities as we can point to the motor and visual zones, but we often can make the topographical diagnosis with sufficient certainty even for surgical purposes by combining various modes of investigation, as: (1) by a few positive localising symptoms; (2) by the general symptoms —such as, in brain tumour, for instance, choked disc, secondary atrophy, headache, vomiting and vertigo; (3) by excluding lesions of the motor, speech, visual and auditory areas, and their association tracts; (4) by special pressure and invasion symptoms—invasion by lesions growing from adjacent areas to those under consideration.

Lesions of the pre-frontal lobe, although this is one of the so-called latent districts of the brain, have in a large percentage of the carefully studied cases shown distinctive manifestations. The symptoms are largely psychical, and unfortunately the physician is not usually well trained to study such phenomena. Mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason; closely studied, the inhibitory influence of the brain both upon psychical and physical action is found to be diminished. Memory is not seriously affected although a continuous train of thought cannot well be followed, and complex intellectual processes cannot be thoroughly performed. The results of experiments upon lower animals have not been very helpful towards determining the

Bechterew[1] maintains with reason that the loss of sensation in animals who have had the motor area of the convolutions destroyed is apparent and not real; that they cannot withdraw the irritated extremity, though they feel the pain, because they have no control of the muscles. He also considers the loss of the muscle sense only apparent, because if the animal's paws be placed in an uncomfortable position its failure to be removed is due to motor inability rather than impaired muscle sense. The animal will move away if irritated on the affected limb, thus showing that it feels but cannot withdraw the limb except by moving the entire body. He concludes that tactile sensation is behind and external to the motor area, and the centre for muscle sense and pain is at the beginning of the fossa of Sylvius.

In several cases of surgical operation on lesions of the motor zone, it has been claimed that the presence of sensory symptoms supports the opinion that the motor areas subserve a sensory as well as a motor function; the case of Macewen,[2] for example, of proto-spasm of the hallux preceded by sensory impressions and followed by paralysis, in which a tubercular nodule was found in the upper part of the post-central gyre. Such a case may prove contiguity but not coincidence of motor and sensory areas.

The neurologist and surgeon must therefore depend on motor symptoms alone in fixing the site for operation in cases in which motor symptoms are definite. When positive sensory symptoms are present, they should regard these as indicative of the extension of the lesion towards either the limbic lobe or the posterior parietal convolutions; or the involvement of the fibres going or coming from these gyres in the corona radiata.

We have no clinical or pathological data with reference to the so-called muscular sense of positive practical value to the physician or surgeon. The hypothesis that the cortical motor zone is rather a zone for muscular sense has little to support it either in physiology or in clinico-pathology. I can see no reason for requiring a muscular sense entirely distinct from other acknowledged forms of sensibility. Some facts have been brought forward which are supposed to indicate that a separate cortical area for registering impressions of muscular sense exists in the parietal lobe behind the motor zone, probably in the inferior parietal lobule; but these observations are not convincing; they certainly cannot yet be made of value in topographical diagnosis for surgical purposes.

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[1] *Neurol. Centralbl.*, Leipz., 1883, ii., 409-414.

[2] *Lancet*, Lond., August 11, 1888.

carefully or the results, particularly for tactile sensibility, are worthless. Patients cannot often be depended upon, as with reference to such tests the greatest differences in the personal equation are found. Some patients have a fashion of stopping to think or weigh their answers when being tested for cutaneous sensation. This often renders the answers worthless; the response should be given promptly.

One of the commonest methods of investigating cutaneous sensibility is that of Weber, which depends upon the fact, or the alleged fact, that the distance apart at which two points can be discriminated is much the same for the same local areas in different individuals. Tables have been published which are supposed to show the least normal distances at which two points can be distinguished. This method, if very carefully applied, is sufficiently correct for many cases, but often the results obtained from such testing are utterly worthless. I have known not only patients with real or alleged loss of sensation, but perfectly healthy individuals to give diverse and confusing answers when tested with the æsthesiometer. Some of the records of the presence of anæsthesia or analgesia in reported cases have doubtless been made improperly as the result of carelessly testing the power of the patients to discriminate between two blunt or sharp points.

Another source of error, particularly in recording cases, is in deciding that objective insensibility exists from the statements of the patients with reference to their feelings. Whatever may be the explanation, it is true that many, though by no means all patients suffering from motor paralysis the result of a cortical or sub-cortical lesion, complain of certain paræsthesias. Spontaneously or in response to questioning they will refer to a paralysed member, or a portion of it, as feeling heavy or numb, or cold, or as if asleep; but often in these cases careful and elaborate testing for true depression or loss of sensation will fail to reveal its presence in the slightest degree. I have been for some time engaged in minutely studying cases of hemiplegia, monoplegia, and aphasia with a view of determining in the most detailed manner the exact movements or forms of speech impaired or abolished, and also whether or not in these cases localised defects of sensation were present. The investigation is a clinical study in localisation, the value of which will be much enhanced by the number of autopsies obtained in the cases studied, but even without such autopsies some results both curious and valuable can be obtained. A single case will serve to illustrate the point which I wish to make about some sensory investigations tinnitus, beclouded mind, slight amnesic aphasia and colour blindness, the patient not being able to tell green from red, although his field of vision was not limited. A triangular focus of softening was found in the left cuneus, most of the cuneus being involved.

Fortunately I had the advantage a few hours before delivering this address of hearing one of the best papers yet written on the cortical localisation of cutaneous sensations, read by Dr. C. L. Dana before the American Neurological Association, and since published in the Journal of Nervous and Mental Disease for October, 1888. Dr. Dana discusses the three views with reference to this localisation: (1) that the centres for these senses are entirely separate from motor areas; (2) that the cutaneous sensory centres are more or less identified with the motor centres; and (3) that both the motor zone and the limbic lobe are concerned in the representation of cutaneous sensation. His paper deals principally with pathological and clinical data. Altogether he collected 142 cases, including four personal observations. He concludes, among other things, that the clinical and pathological evidence collected by him shows that the motor areas of the cortex contain also the representation of cutaneous sensations; and that cutaneous anæsthesia of organic central origin is always limited to or more pronounced in certain parts. The same objections apply to most of his cases as indicated for those of Starr.

Dana collected for this paper twenty cases in which the gyrus fornicatus or hippocampal convolution were more or less involved, and in none of these, he says, was any anæsthesia present that could be fairly attributed to the lesion. Even a casual study of these cases seems to me to show their weakness for the support of the position taken that the limbic or falciform lobe is not concerned with sensation. In nearly one-third of these cases some disturbance of sensation was present. Three of the remaining cases were demented, and presumably any investigation into cutaneous sensation would have but little value; one case presented no symptoms.

What Ferrier says of defective methods of investigation and erroneous interpretation of the reaction of the lower animals to sensory stimulation may be applied with some additions to the study of cutaneous sensation in cases of cerebral disease in man. Numerous sources of error are present, not only in investigating but also in reporting the results of investigation.

To test cutaneous sensibility requires time, patience, and method. It is often tedious and monotonous; but it must be done extremities or the face. This fact is not to be explained as simply from irritation of the pia mater; for on the other hand I have observed cases of localised lepto-meningitis in which both hyperæsthesia and even pain in the head were absent. Such cases also cannot be fully explained by tubercular deposit in and inflammation of the dura mater. Although dural meningitis may cause headache of great severity and sensory phenomena in the domain of the fifth nerve, it should not be held responsible for hyperæsthesia of the extremities. The hyperæsthesia in these cases is best explained by irritation of sensory cortical areas.

Bernhardt[1] has recorded the case of a woman, forty-one years of age, who after an apoplectiform attack had hyperæsthesia of both lower extremities; also paresis of the right arm, lower face and foot; spasm involving the right hand, arm and face; weak reflexes of the right leg; confusion, excitability and aphasia. The lesions were a tubercular growth in the upper extremity, lateral and median portion of the left posterior central convolution and in the precuneus; a similar growth in the middle portion of the posterior central gyre; and a tubercle the size of a pea at the anterior extremity of the corpus striatum. The hyperæsthesia in the lower extremities might be explained by the tumour in the great median fissure. Hitzig[2] quotes from Löffler the account of a soldier who received a gunshot wound on the top of the head, the dura mater being pressed inward by the depressed bone at a point which corresponded with the upper ends of both central gyres. Both legs were hyperæsthetic and also paralysed. Such symptoms can be explained by pressure on the paracentral gyres of both sides and direct or inflammatory irritation of the neighbouring sensory cortex below or behind.

Bramwell[3] has reported a case of incomplete paralysis of the left extremities and of the face, with disturbance of sensation of both the arm and leg; with also atrophy of both optic nerves and other sight disturbances. The autopsy revealed a tumour with softening of the surrounding brain in the right superior and middle parietal convolutions and a part of the first occipital convolution.

Brill[4] records the history of a patient with right-sided anæsthesia and paresis, the former more marked than the latter. In a few weeks hyperæsthesia developed, with also restlessness, these cannot, however, overcome positive evidence of decided destructive lesions of the cortical motor centres, without any disturbance of touch, pain or temperature, or even of the muscular sense; and a close study of the cases of cortical lesion in which both motor and decided sensory phenomena have been present will not bear out fully the view of Starr.

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[1] *Arch. f. Psych.*, iv.. heft 3, 1874.
[2] *Op. Cit.*
[3] *Lancet*, September 4, 1875; ii., 346.
[4] *N. Y. Med.* vol. xxii., 1882, July 15, 81-82.

One of Starr's tables contains forty-one cases showing cortical lesions of the brain with sensory symptoms. That the sensory area lies about the motor region might even from these cases be conceded, but that the two coincide has been by no means proved, particularly if the postero-parietal region is not regarded as a necessary part of the motor area. Reviewing the forty-one cases, in only one-fifth to one-sixth are the lesions restricted to parts of the brain in front of the central fissure, although in many of the others both the anterior central and posterior central gyres are involved. In more than thirty of these cases the reported lesions involved in part or whole the postero-parietal region and posterior central gyre. Four of my cases are included in the table, and this has led me to look over not only these but also other of my cases of cortical lesion with sensory phenomena; and from doing this the conclusion has been reached that they do not support the doctrine that sensory and motor areas entirely coincide.

In the case of a fibroma which involved the first and second frontal gyres and anterior segment of the gyrus fornicatus and corpus callosum, anæsthesia of the conjunctiva, with conjunctivitis and corneitis were present. In a patient with a gumma involving the upper fourth of the pre-central and a smaller segment of the post-central gyre, hyperæsthesia was marked, with also crural Jacksonian epilepsy, but the lesion grazed the sensory area. A carcinoma of the middle portion of the posterior central gyre and the upper part of the inferior parietal lobule was accompanied with wide-spread destruction of the corona radiata, and impaired sensibility of the limbs of the opposite side with other phenomena was present. Owing to the posterior extension of this growth and the large sub-cortical destruction, this lesion could certainly not be claimed to teach anything with reference to the coincidence of motor and sensory areas. In another case of tubercular tumour in the motor area, the interior of the hemisphere was broken down, and hemianæsthesia, at first partial but later complete and persistent, was present.

Numerous observations have shown me that hyperæsthesia is a common phenomenon in cases of diffused cortical tubercular meningitis, and this hyperæsthesia may involve any or all the case, complicated by the presence of a certain amount of motor paralysis, chiefly, if not entirely, affecting the muscles of the leg. We have no doubt that this condition was always due to a lesion (accidentally produced during the operation, or subsequently as the result of interference with the circulation) in the leg area of the marginal convolution. Now in one or two of these cases the anæsthesia affected chiefly the paresed limb, and it might be argued by those who, like Schiff and Munk, hold that the excitable areas of the cortex are concerned with the perceptions of sensory impressions from the corresponding regions of the body that the loss of sensibility was due to lesion of the motor area. But against this argument we may not only put forward those experiments in which there has been no accompanying paralysis, but also others in which the hemianæsthesia has been well marked in the upper limb and upper part of the trunk, while the lower (paresed) limb has exhibited no diminution of sensibility whatever. . . . . .

"From which it would appear that while the whole limbic lobe may be concerned in the perception of sensory impressions, the part played by the gyrus fornicatus, at least as regards cutaneous sensibility, is more important than that played by the hippocampal portion of the lobe. But until it has been possible entirely to destroy the whole lobe upon both sides of the brain it is impossible to determine whether it is assisted in its functions by any other portion of the cerebral cortex. . . . .

"The results of our experiments upon the limbic lobe seem to point to the conclusion that this portion of the cerebral cortex is largely, if not exclusively, concerned in the appreciation of sensations, painful and tactile. This is an extension of the view put forward by Ferrier, who was inclined as the result of his own experiments to limit that function to the hippocampal region. Dr. Ferrier was good enough to assist at some of our experiments upon this part, and has fully accepted the conclusions to which they point."

Starr, from a series of American cases of cortical lesions of the brain,[1] and also from a study of the sensory tracts,[2] concluded that the various sensory areas lie about and coincide to some extent with the various motor areas for similar parts; that, in other words, the Rolandic region is a sensory-motor region, the sensory area, however, including to some extent the gyres of the adjacent postero-parietal lobe. Collections of cases such as observers like Schiff, Munk and others who believe that sensory areas coincide with motor centres, is reasonable. "The conclusion that tactile sensibility is lost or diminished after the destruction of the cortical motor area," he says, "is based on defective methods of investigation and erroneous interpretation of the reactions of the lower animals to sensory stimulation. Though an animal does not react so readily to sensory stimulation of the paralysed side it does not follow that this is due to diminished or absent perception of the stimulus. An animal may not react, or react less energetically, to a sensory stimulus, not because it does not feel it the less, but because it is unable or less able, to do so from motor defect. It is astonishing what apathy or indifference some animals display towards certain forms of stimulation, such as gradually increasing pressure on the fingers or toes which one would regard as well calculated to elicit reaction or signs of uneasiness. Unless the stimulus is of a nature to at once excite attention, or to evoke reflex action, it may appear to be altogether unperceived. All that the experiments of Schiff and Tripier demonstrate is that motor reactions are less readily evoked on the side opposite the cortical lesion. But the same thing occurs in cases of purely motor hemiplegia in man."

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[1] *Am. Jour. Med. Sci.*, Phila., 1884, n. s., lxxxvii., 366-391.

[2] *Jour. Nerv. & Ment. Dis.*, vol. xi., No. 3, July, 1884, 327-407.

On anatomical and morphological grounds Broca holds that the callosal and hippocampal regions constitute together a distinct lobe of the brain, the falciform or limbic lobe; also I believe erroneously, that this lobe is connected entirely with the sense of smell. So far as the brain of man is concerned, in this lobe should probably be included the entire precuneus or quadrate lobule on the mesial aspect of the hemisphere and the adjoining portion of the superior parietal convolution on the lateral aspect of the hemisphere.

The most important recent contribution to cortical sensory areas is certainly that of Horsley and Schäfer.[1] Hemianæsthesia partial or complete and contra-lateral resulted from destructive lesions of the limbic lobe. They found that any extensive lesion of the gyrus fornicatus was followed by hemianæsthesia more or less marked or persistent; sometimes the loss of sensation involved almost the whole of the opposite side of the body; sometimes it was localised either to the upper or lower limb, or to a particular part of the trunk. They did not, however, succeed in establishing the relations between special regions of the body and the parts of the convolutions which had been destroyed.

"These experiments were frequently, but by no means in every sensations of touch, pain, temperature, and muscular sense, and lesions in its course will cause disturbance of these sensations. Like lesions in the motor tract, the rule obtains that the nearer the cortex the more likely is the lesion to cause an affection of a single limb, while the nearer the capsule the more likely is the symptom produced to be hemianæsthesia."

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[1] Phil. Trans. Royal Soc., 1888.

Ferrier in his earlier experiments found that lesions involving the horn of ammon and hippocampal convolution, caused impairment or abolition of tactile sensibility on the opposite side of the body, and located the cortical centres for this form of sensibility in the hippocampal region. In experiments with Professor Yeo, in 1884, he established lesions in the hippocampal regions in ten monkeys, and in five of these in both hemispheres, and these lesions showed that tactile sensibility was in every case impaired or abolished, according to the amount of destruction of the hippo[illegible]ampal and temporo-sphenoidal region. It was also established, [illegible]owever, that a very extensive lesion might be made in one or [illegible]oth hippocampal regions without producing permanent anæs[illegible]esia.

Munk[1] claims that the area of common sensation—including sensation of pressure, location of a limb, muscular sense, and touch—lies in the central region, including the anterior and posterior convolutions and adjacent cortex. He divides this area into special centres for the hind leg, fore leg, head, eye and ear muscles, neck and body; holding that these coincide with but are somewhat more extensive than the corresponding motor centres for these parts.

The sensations of touch, according to Luciani, are perceived in the central convolutions, and therefore lesion of these cause anæsthesia as well as paralysis. The tactile area includes the parietal convolutions also, but does not reach the occipital and temporal lobes. This author holds that all sensations appear to have a common zone in the parietal region, and lesions in this common zone may cause disturbance of all the senses.

It is worthy of remark that the physiologists and others, who contend that the sensory portion of the cortex coincides with the motor, also hold that it extends beyond into adjacent cortical regions, as witness the views of Munk, Tripier, Moeli, and others cited by Starr in his elaborate paper.

Ferrier's explanations[2] of what he regards as the errors of had a fit or seizure, the character of which was not closely observed. Afterwards, however, she was paralysed and anæsthetic over the right half of the body and limbs, her speech also being thick, but consciousness not lost. Partial anæsthesia was present over the left half of the body. Knee-jerk was increased, and ankle clonus was present on both sides. She improved rapidly, but remained in bed two weeks. Two days before her death she had an apoplectic attack, and became completely paralysed and hemianæsthetic in the face, limbs, and trunk of the left side. The autopsy showed a foyer of softening in the right hemisphere, including the entire breadth of the internal capsule, two-thirds of the adjoining lenticular nucleus, a small anterior external portion of the thalamus, and a narrow strip of the caudate nucleus where it curves around the thalamus. Careful transverse sections showed that the anterior boundary of this focus of softening was one and a-quarter inches behind the head of the caudate and lenticular bodies, and that it extended from backwards about five-eighths of an inch. It was so situated that a transverse section immediately in front of the thalamus marked its anterior boundary. The softening of the narrowed caudate nucleus was all that was observable in opening the ventricle. The left hemiplegia and hemianæsthesia might very properly be attributed to the complete transverse destruction of the internal capsule. A large recent embolus was found in the right middle cerebral artery. It is difficult to account for the temporary right hemiplegia and hemianæsthesia, except on the theory of hysteria or functional disturbance, as no lesion of the cerebral hemisphere on the left side was discovered.

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[1] *Pfluger's Arch. f. Physiologie.* Quoted by Starr, *Jour. Nerv. and Ment. Dis.*, vol. xi., No. 3, July, 1884, 327-407.

[2] 'Functions of the Brain.'

The fibres of the sensory tract are probably separate from the motor even in the centrum ovale.

"The third set of fibres of the projection system," says Starr,[1] "includes those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions. These take a similar course to the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, towards the occipital lobe. They are mingled with fibres which pass to the optic thalamus, but are separable from them, as Edinger has shown, in fœtal brains, and may be traced down through the capsule to the tegmentum of the crus, where they divide into a portion going to the leminscus, and a portion going to the formatio reticularis. This set of fibres conveys the cortical areas. Fournier believes that sensory fibres terminate in the thalamus. Crichton-Browne regards this ganglion as a great centre of general sensibility: but Flourens, Longet, Schiff, and Tamburini, among others, have assigned to it motor functions. The weight of the evidence, both from experiment and disease, connects the thalamus with various forms of sensation. It is probably a halting place where sensory impressions going from the periphery to the cortex undergo some transformation. It is necessary, at least, briefly to thus refer the functions of the thalamus as some authorities are inclined to regard it as the sole cerebral region for sensibility.

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[1] *Medical Record*, Feb. 13, 1886.

From analogy and general principles it is altogether probable that we have above the ganglia a separate localisation for the different forms of cutaneous sensibility. As Ferrier puts it, up to the point of radiation towards the cerebral cortex the sensory paths have been proved to be entirely differentiated from the motor, and it is therefore extremely unlikely that the two should become jumbled together indiscriminately in the cortical centres. We have not only the well known differentiation between sensory and motor nerve roots and tracts in the spinal cord, oblongata, pons, and crura cerebri; but also abundant [illegible] as to the separation of sensory from [illegible] capsule. The very simple but nat[illegible] re do these paths lead if not to [illegible]? Cases have been reported [illegible] F. Müller and others, in [illegible] motor [illegible] sthesia to result from lateral [illegible] psule or trench backwards and upwards nearly to the sagittal fissure, crossing and confluent with the interparietal. In a Chinese brain studied by Dr. A. J. Parker and myself, a remarkable peculiarity of the brain was that in the right hemisphere the first temporal fissure, beginning near the anterior extremity of the temporal lobe, passed backward across the entire extent of the parietal lobe and over the median edge of the hemisphere for the distance of half an inch, terminating in the precuneus just in front of the parieto-occipital fissure.

This is an interesting field, and has some practical importance. Even the Sylvian, the central, and the parieto-occipital fissures have considerable variations; the first in its horizontal level and extension, and the others in their antero-posterior position. I have reported one case in which the Sylvian fissure took a position much more vertical than usual and reached nearly to the longitudinal fissure. As a rule, variations, such as those referred to here, for fissures like the Sylvian, woul not confu se us in operating after the methods of Broca, Thane, Reid, Horsl ey, and others; but they are certainly worthy of some consideration n.

Locali

With referenc es for cutaneous sensations—tou sensations are acquiring more positiv ne to use such knowledge lence, physiological, ger in favour rants, and probably even smaller portions of the retinal expansion are related to separate areas in the brain. The macular region almost certainly has its special cortical centre. The tendency of investigation, both experimental and pathological, is to show that the whole of the occipital lobe, and adjoining portions of the parietal and temporal lobes, are in some way concerned with vision, as I have tried to indicate in the diagram.

With reference to visual localisation, the importance of confirming physiological experimentation by careful clinico-pathological observations cannot be over-estimated; these observations are here even of more importance than in motor localisation. The movements of a limb or a portion of a limb can be studied with considerable accuracy in the lower animals as well as in man; but in investigating sight, the other special senses, or general sensibility, in the lower animals, we are confronted with special difficulties and sources of error. This is perhaps more strikingly true of hearing, taste and smell than of the other senses, but it is true for all. Schäfer, referring to a monkey from which he believed he had eradicated both angular gyres, states that for three or four weeks the animal failed to see objects which were just below or to one side of its eyes, and even at the time of writing there appeared to be complete absence of vision in the antero-superior and lateral portions of the retinæ; but it was difficult to prove this, because the monkey had acquired the habit of rapidly directing his head and eyes so as to use the central parts of the retinæ.

Operations guided by Visual Localisation.

In spite of the conflict of views with reference to visual localisation, sufficient has been determined to indicate several positions for operation guided chiefly by visual symptoms.

If a patient has lateral homonymous hemianopsia as the special localising symptom, operation should be performed with the view of reaching the cuneus behind the position of the parieto-occipital fissure. If, without hemianopsia, the patient fails in intelligent recognition of things and words, the aim should be the lateral occipital convolutions and the angular gyre, which is adjacent, or indeed may be situated in the occipital lobe. When, with lateral homonymous hemianopsia, the patient also has hemianæsthesia, the lesion is probably in the tracts between the cuneus and the primary optic centres, large enough also to involve the sensory tracts. Such a lesion would probably best be reached

In illustration of the effects produced by complete removal of the occipital lobe, and of that alone Schäfer gives two instances, in one of which the operation was unilateral, in the other bilateral.

"In the monkey upon which the unilateral operation was performed, the left occipital lobe was removed by a vertical incision carried along the line of the parieto-occipital fissure. That the removal was exact and complete was confirmed on *post-mortem* examination, some eight months after the establishment of the lesion, when it was seen that the whole of the occipital lobe, and only this lobe was involved, the angular gyrus being quite intact and normal, and the surface of the section looking as fresh, and showing as clearly the distinction of grey and white matter, as if the operation had just been performed. The result was the immediate establishment of bilateral homonymous hemianopsia, which persisted the whole time the monkey was kept alive. Objects so placed that their images fell upon the left half of the retinæ were taken no notice of: a threatened blow coming from the right-hand side of the mesial visual plane was winced at or avoided; currants strewn upon the floor were only picked up towards the left side, the animal working in that direction. In the case of the monkey with bilateral operation the result was total and persistent blindness."

The views of Hun[1] are that the convex surface of the occipital lobe, particularly of the left side, is associated with complete visual perception and recognition; and also that the so-called left angular gyre is essential for the memory of the appearance of words, lesions of it causing alexia and agraphia.

I shall not attempt to analyse, criticise, or reconcile these various and varying views. They agree at least in showing the production of hemianopsia from lesions of the cuneus and adjacent occipital lobe.

The general visual zone which has been determined can probably be compared as to subdivision with the general motor zone. As the motor zone has been subdivided into areas of representation, not only for the leg, trunk, arm, face and speech, &c., but also into areas or centres for parts of the leg, arm, face and speech, so efforts partly successful are now being made to subdivide the visual zone. The retina, so far as its connection with the central cortex is concerned, can be subdivided into segments probably of a somewhat regular shape. At first, studies in hemiopia and hemianopsia seemed to show that the only definite connection was between the halves of the retinæ and cortical centres, but the latest observations indicate that quad-on which he was engaged with Mr. Victor Horsley during more than two years, Schäfer[1] writes as follows:

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[1] *Op. Cit.*

"With regard to vision our experiments were not conclusive. We found that extensive lesions, both of the occipital lobe and of the temporal lobe, were invariably followed by visual disturbances, taking the form, when the operation was confined to one side of the brain, of bilateral homonymous hemianopsia; but in nearly every case the hemianopsia was merely temporal, and after a certain time we could not in our monkeys obtain any distinct evidence of the persistence of the visual defect. The most marked results of this kind were obtained when the occipital lobes were the seat of operation, extensive unilateral lesions in this region producing hemiopia, and bilateral lesions producing amblyopia; but in neither case were the symptoms permanent, and after a time the animals, so far as we were able to determine, could see as well as their intact fellows. In one case only did the hemiopia persist, and this was one in which, after a bilateral lesion of both occipital lobes had been carried out and the temporary blindness thereby produced had been recovered from, the angular gyrus of one side was destroyed. This second operation, made upon the animal in which the occipitals had already been extensively destroyed *without* permanent blindness, did produce a condition of hemianopsia which lasted until the animal's death some three months later. We were of opinion at the time that this instance might warrant us in taking up a position similar to that of Luciani and Tamburini, and intermediate between those of Ferrier and Munk—the former of whom originally denied the participation of the occipital lobe in the visual perceptive function, and still appears to regard it as subordinate to the angular gyrus; whereas the latter would localise those perceptions entirely in the occipital lobe and deny all participation of the angular gyrus. But we made only four experiments upon these regions, and in none of them was the removal of the occipital lobe complete, as was proved by *post-mortem* examinations of the brains. They were not, therefore, decisive against Munk's statement, that persistent hemiopia or blindness follows extirpation of one or both occipital lobes alone, and it became necessary to pursue further inquiries in order to test its accuracy."

In conjunction with Dr. Sanger-Brown, Schäfer also experimented upon the angular gyre and the occipital lobes. Destroying one angular gyrus as completely as possible with the actual cautery they could discover no defect of vision, no loss of movement of the eyes or eyelids, and no anæsthesia of the corneal conjunctiva. A week later the angular gyre of the opposite side was destroyed, also with negative results.

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[1] *Brain*, London, January, 1888.

During the last month of his life he vomited often. He complained of increasing dulness of vision and of greater angle of obliteration, and was much troubled by a new building near by appearing to be out of line. On May 7, 1886, while quietly walking in the street, he sank gently to the ground and died.

At a point on the median surface of the right occipital lobe was complete atrophy of the cerebral convolutions, only a trace of them remaining as a delicate gray gelatinous fringe. This atrophy was strictly limited to the lower half of the cuneus; being bounded below by the calcarine fissure, in front by the parieto-occipital and above by a curved line which started from the parieto-occipital fissure, and arching backwards across the middle of the cuneus terminated at the posterior border of the median surface close to the calcarine fissure. The white matter underneath the point of atrophy was softened to a depth of about one-third of an inch. There was no deposit of pigment in the neighbourhood. The corresponding point on the left occipital lobe showed no atrophy, nor did any of the other cerebral convolutions. Sections through the brain substance, the optic thalami, and the other ganglia at the base revealed nothing abnormal. The optic nerves and tracts showed no microscopic atrophy or degeneration. No microscopical examination was made.[1]

Views of Physiologists as to Visual Localisation.

The view of Ferrier as expressed in the latest edition of his 'Functions of the Brain,' is that the angular gyres maintain relations with the areas of clear vision, and as a matter of course especially with the macula luteæ.

This physiologist has modified his earlier views in so far that he no longer localises the visual centres in the angular gyres to the exclusion of the occipital lobe; but believes now that the visual centres embrace not only the angular gyres, but also the occipital lobes, which together he terms the occipito-angular regions.

Recently considerable activity has been exhibited in the investigation of visual areas.

From a long series of experiments upon the monkey's brain, examined this man in 1885, he presented no distinct paralysis, no anæsthesia, no aphasic symptoms. His tongue deviated a little to the right, and the grasp of the right hand was a little lighter than that of the left. A rough test with a small white object at eighteen inches showed right lateral hemianopsia, with a line passing outside of point of fixation, and a darkened area in the left upper temporal quadrant. Pupillary reaction was normal. Examination of fundus showed blood vessels of normal size; outer temporal quadrant of each disc whiter than normal; left a little whiter than the right. An experiment on the cadaver showed that the track of the ball was such that it must have injured the optic fasciculus on its way to the cuneus.

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[1] At a recent meeting of the Philadelphia Pathological Society, I presented the brain of a man who had been blind more than twenty-five years—how much more could not be positively ascertained. Both occipital lobes were unquestionably small. The cuneus on each side was small, the first occipital convolution of Ecker (superior external *pli de passage* of Gratiolet, and par-occipital of Wilder) showing lack or arrest of development. The second and third occipital convolutions of Ecker, especially on the left, presented a narrow, dwindled appearance. In another brain of an old woman, blind for at least thirty years, similar gross appearances of arrested development in the occipital region were present.

The most important case which has been recorded since the paper of Seguin is that of Hun, in which a defect in the fields of vision involving the lower left quadrant of each eye occurred with atrophy of the lower half of the right cuneus.

The patient, a man aged fifty-seven, in 1869 had a severe attack of double pneumonia, and during the year following slight attacks of vertigo while walking, which were attributed to weakness. From 1877 until his death, he was troubled by slight deafness and by more or less roaring in his ears. In 1882 he had a large carbuncle on his neck, and after that time he seemed less vigorous than before. Early in December, 1884, he was examined by Dr. Merrill, who found normal reaction of the pupils, normal appearance of the fundus, vision and colour perception perfect, but a defect in the fields of vision involving almost the whole of the left lower and the peripheral portion of the left upper quadrants in each field. The defect was somewhat more extensive, especially as regards the upper quadrant, in the left field of vision than in the right.

From this time the condition of the patient did not change materially. He continued to be very nervous, and at times irritable and suspicious. He exhibited no paralysis of motion or sensation. His memory was weak in regard to names; he often called the same person by several names in the course of conversation; while in other respects, as in recognising faces, his memory was excellent. He slept but little, and in his sleep there was much twitching of the limbs. In 1885 he had a severe attack of angina pectoris, and for two weeks he could walk only a short distance without bringing on an attack of the pain. In February, 1886, he had his most severe attack of angina, lasting several hours, and from this attack until his death he was scarcely a day without pain in the precordia or in the arm.

was found on autopsy to be a large focus of yellow softening, evidently an old patch, involving the basal part of the cuneus, the fourth and fifth temporal convolutions of Ecker, and a part of the hippocampal gyre. Other lesions were present in the brain, but as this case was in harmony with others Seguin regards it very properly as of great value.

In his table in the paper referred to on cortex hemianopsia, Seguin has included five cases of traumatic hemianopsia due to injuries of the occipital region of the skull, and lesion of the subjacent brain. I will refer to only one of these cases, that of Keen and Thomson, which is of historical as well as of scientific interest. Through the courtesy of Dr. Keen, I have examined a cast of the head of this patient. The case was reported in the photographic *Review of Medicine and Surgery*, February, 1871; also in the 'Medical and Surgical History of the War of the Rebellion,' Part 1, p. 206-207. It is also referred to in Flint's 'Physiology of Man,' vol. v., p. 41-42, 1874, as one of the first cases to show that the filaments from the optic tracts on the two sides are connected with distinct portions of the retina. The patient was kept under observation off and on for a number of years, and was examined and exhibited by Dr. Seguin at the time of the reading of his paper. His hemianopsia was found unchanged twenty-three years after the reception of his injury. The patient died within a year or two, but unfortunately no autopsy was secured. He was hit on the head by a Minie ball at the battle of Antietam, September 17, 1862. The wound of entrance was in the middle line one and a quarter inch above the external occipital protuberance; it made its exit about two inches to the left of the middle line, and three above the wound of entrance. He noticed impairment of vision two days after the injury. About ten days afterwards he had an attack of loss of consciousness with some paralysis of the right arm and right leg, which lasted some two or three months. His memory was imperfect for some months, but he had no aphasia. In walking he was very giddy, and noise and laughter would hurt him. His mental and physical powers gradually grew better. He was seen and examined by Drs. Keen and Thomson, in December, 1870. Among other conditions he was found to have a complete lateral hemianopsia. Upon testing the field of vision it was found to be divided in each eye by a line passing through its centre in a vertical direction—total blindness existing to the right, and perfect vision to the left of this line. Ophthalmoscopic examination showed no pathological appearances. When Dr. Seguin

It was noticed after some months that the patient held her head obliquely to the left. Examination revealed left homonymous hemianopsia, the only symptom indicating a focal lesion of the brain. The patient died of broncho-pneumonia. At the autopsy two tumours were found in the brain; one at the apex of the left frontal lobe. The second tumour lay in the mesial aspect of the right occipital lobe, projecting a few mm. above the level of the brain, firmly adherent to the pia and only slightly to the dura. Its length was 3 cm., height 3 cm., thickness 2.5 cm.—mostly buried in brain substance. It lay directly over the sulcus hippocampi, extending to either side of it. The base of the occipital lobe was not involved.

Féré reported the case of a female, aged fifty-two years, who in November, 1883, had a sudden apoplectic attack followed by right hemiplegia. She had partial and slight right hemianæsthesia to cold and pain. Hearing, taste and smell were normal; and typical right lateral hemianopsia, the vertical line passing through the point of fixation, was present. Autopsy showed a yellow patch destroying the greater part of the left cuneus and encroaching somewhat on the adjacent occipito-temporal convolution, the fifth temporal convolution of Ecker. No other lesions were present.

Seguin reports one personal case. The patient, a man forty-six years old, consulted him first in January, 1884, for insomnia and dyspepsia. He was treated for various symptoms with varying success. December 5th, 1884, he had an attack in which he complained of numbness in the left cheek, arm and face, and most marked in the hand and foot. He had no distinct hemiplegia, and no hemianæsthesia, but thought that tactile sensibility as in passing his fingers over objects was somewhat duller. He could not see objects on his turning his head and eyes, and testing revealed left lateral hemianopsia, with a vertical division line not including the point of fixation. Central vision was good. He continued with varying symptoms until May 17, 1885, when he died. During his long illness he had attacks of acute hallucinatory mania, both aural and hallucinatory illusions being present. He had chills, high fever, and sweats which followed no distinct periodicity. Previous to his death his speech was sometimes somewhat difficult to understand. The hands both showed disorders of movement, choreiform tremors, and in the left hand slight ataxia and larger motion. The hemianopsia persisted to the last unchanged. The central vision remained good. The brain lesion which doubtless caused the hemianopsia

Jastrowitz has recorded a case of paresis of the right leg, arm and face, with a peculiar form of aphasia. The patient was unable to read and write connectedly; he could not understand written words. The history does not relate definitely whether or not he understood spoken words. Right hemianopsia was also present. The autopsy showed tumour of the left occipital lobe and precuneus.

Seguin, in a contribution to the pathology of hemianopsia of central origin, in many respects the most valuable publication on the subject which has yet appeared, collected forty cases with autopsies, and five traumatic cases without autopsies. Eleven of these were cases of hemianopsia due to lesions of the white substance of the occipital lobe. Sixteen were cases of cortical lesion, or of lesion limited to the cortex and the white substance immediately subjacent; and four of the sixteen (those of Haab, Huguenin, Féré, and Seguin referred to above) are what might be termed conclusive cases as to the question of the location of at least a portion of the cortical visual centre in man; as in them the lesion was circumscribed and occupied nearly the same place in the occipital lobe. They would, at least, seem to settle definitely that in the cuneus and its immediate neighbourhood the visual half centre for retinal sensations is located. Owing to their importance I will briefly give from Seguin a condensed abstract of these cases.

Haab's case was a man, sixty-eight years old, who had an attack of temporary paresis of the left extremities. The patient complained that he could not see to his left with his left eye—though his right eye was normal. No anæsthesia; intelligence was normal, hearing good. Central vision—1 (H.2). There was left homonymous hemianopsia, the limit reaching quite (?) up to fixation-point. In the right fields colour perception was good. Optic nerves presented a "senile greyish colour." The autopsy showed the extremity of the right hemisphere five mm. shorter than its fellow and a depression in the right occipital lobe, the pia hanging loosely over a cavity containing clear fluid. The patch was mostly upon the mesial aspect of the hemisphere (including apex). It occupied the site of the fissure hippocampi, and extended beyond it above and below. The white substance was but slightly injured.

Huguenin recorded the case of a girl, aged eight years, whose chief symptoms were headache in paroxysms; later, frequent vomiting, sleep broken; severe convulsions which frequently recurred; increasing dementia; slight neuritis with some swelling.

cupping and venous pulsation; no arterial movement observable. Slight choroidal change in pigmentation to the temporal side of the nerve; outline of nerve above not defined, but merged with the retina. Macula good. Hypermetropia. *Left eye*:—Pupil more dilated and less active than in the right eye. Appearances much like those seen in the other eye, but worse. Evidences in colour and margin of slow chronic inflammation. Macula good. Right eye, vision, $\frac{20}{50}$; left eye, vision, $\frac{20}{70}$.

After her illness had continued several months, the patient began to experience at times attacks of flexure of the fingers and thumb of the left hand, and cramp-like feelings in the forearm and arm. Later these spastic attacks began in the left foot, causing the great toe to be flexed upward and the other toes downward, with also some cramp feelings in the left leg.

A number of consultations were held with reference to this patient with Drs. J. B. Roberts and C. B. Nancrede, who took the view that the case was probably hysterical, and treatment was eventually suspended.

The patient's general health improved; she gained in weight and attended to household and business affairs, never however getting entirely rid of her main symptoms, and recently some of these have returned with renewed vigour. She has fallen several times in public places and at her own home, appearing to become entirely unconscious. The attacks are preceded by a thrill passing up the left leg, and twitching of the left arm; the latter continuing through the entire seizure and being the only muscular movement noticed.

Visual Localisation.

Next to determinations of the motor zone visual localisations are the most conclusive, and this in spite of the hard-fought battles of the physiologists over the cortical sight areas. Clinical medicine and pathology have here come bravely forward to clear away the storm. A few well-reported cases of hemianopsia with autopsies, as those by Jastrowitz,[1] Haab,[2] Huguenin,[3] Monakow,[4] Seguin,[5] Hun,[6] Féré, Keen and Thomson, seem to settle beyond doubt the connection of the cuneus and adjacent region with the retina and simple visual sensati localisation and extent can be found in the descriptions of hystero-epilepsy. Features of distinction are however present. Undoubtedly one reason for the similarity between spasmodic affections reflex, hysterical, toxic and cerebral, lies in the fact that in these cases, whatever may be the starting point, central areas are discharged and give definite character to the convulsions. Horsley speaks of hystero-epilepsy as a cortical disease, but this view cannot be upheld for all cases, if he means by this that the spasms are usually the result of cortical discharge. They are rather sometimes bulbar or spinal, cortical inhibition being removed.

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[1] *Centralb. für prakt. Augenheilk*, v. 1879, p. 254.

[2] *Klinische Monatsblatter f. Augen.* 82.

[3] *Ibid.*

[4] *Archiv. f. Psychiat, u. Nervenk* 1, S. 166.

[5] *Jour. of Nerv. and Ment. Dis.*, anuary, 1886, 1-38.

[6] *Am. Jour. Med. Sci.*, vol. xciii., 0-168.

The difficulties of making a diagnosis between grave hysteria or hystero-epilepsy, and cerebral tumour or other organic lesion with apparent or real Jacksonian symptoms, is sometimes great, and was strikingly shown in a case seen by me in consultation with Dr. J. M. Barton, of Philadelphia. This patient, a married woman, thirty-five years old, came under observation in the spring of 1886. She had been in bed eight weeks, and had taken no part in her household affairs for several months. Her sickness began with complaints of headaches and feelings of slight numbness in the left hand and arm; she would occasionally drop things from this hand. She soon developed analgesia and anæsthesia in the left arm and leg, and sometimes in the face; this varied in severity. Her mental condition gradually changed, she became irritable, absent-minded, and lacking in attention and judgment. She had at times hallucinations of sight, which usually occurred after lying down, these often taking the form of animals, as cats, mice, &c., disappearing round corners. Hearing was good in the left ear, but in the right was diminished; careful examination showed no external or middle ear disease.

She suffered almost continuously from headache, which she described as violent and agonising, and which she localised mostly in the fronto-parietal region; and a cranial area over the right motor zone was very tender to pressure and percussion. Nausea and severe vomiting came on late, and both headache and vomiting were accompanied by vertiginous sensations. A curious symptom was a constant diar [illegible] tient having from six to fifteen passages a day, and [illegible] s many as twenty-four. She also often spat a bloody [illegible] mouth. Her appetite and sleep were much impaire [illegible] t considerably in weight.

Ophthalmoscopi [illegible] s made in March, 1886, by Dr. W. W. McClure, g [illegible] wing results: *Right eye*:—The pupil more active th [illegible] media clear; slight physiological convulsive movement would extend to the right hand, forearm and arm, and simultaneously the muscles of the lower part of the right side of the face would become affected with spasm, a tremor also appearing in the tongue during this period; fifthly, the right arm and leg would now become affected with a clonic spasm, causing them to assume positions of flexion, the head, neck and body being drawn by the spasm at the same time to the right, a condition of pleurosthotonus being in fact produced. The seizures would pass off with a very severe jerking movement of the right shoulder, and a renewal of the twitchings of the muscles of the right angle of the mouth. These movements of the shoulder and mouth would sometimes occur only once, just before the close of the attack; more frequently, however, they would take place two or three times in succession. Occasionally the patient would bite her tongue during the paroxysms. She apparently was never entirely unconscious during the attack, no matter how severe it might be. During the height of the convulsion, if her hand was pressed too hard she would manage to gasp out, "Don't," or to make some other exclamation.

Unilateral Nervous Affections in Bright's Disease.

Considerable evidence has accumulated to show that affections of the nervous system, strictly limited to one half of the body, occur during the course of some forms of Bright's disease. In this country Dercum[1] has reported cases of hemichorea, hemiplegia and unilateral convulsions. Raymond,[2] Chantmesse and Tenneson[3] reported series of cases of unilateral affections, chiefly hemiplegia and epilepsy apparently of uræmic, or at least renal origin. In not one, according to the reporters, could the trace of a strictly focal lesion be discovered. Chaufford[4] reports a highly-interesting case under the title of uræmic convulsions of the Jacksonian form.

Hystero-Epilepsy and Jacksonian Epilepsy.

Some cases which seem to be clearly forms of hystero-epilepsy closely resemble organic epilepsy of the Jacksonian type. Hystero-epileptic attacks, it is well known, can be produced by irritation of the hystero-epileptogenic zones, described by Charcot, Richer, and others, which are evidently analogous to the epileptogenic zones of Brown-Sequard.[5] Almost every form of spasm in and herein also sometimes is to be found justification for operation for the removal of cortical discharging areas, even when such lesion is not present. Such a method of origination of cortical epilepsy is in accordance with physiological principles. Meynert,[1] in the development of his idea of a projection system, has perhaps more clearly than anyone else made apparent the method in which this result may be brought about. Movements which were originally reflex in character may after a time result from cortical impulses. In the normal brain no reflex actions can be performed without exciting to action secondary volitional movements which no longer requires the stimulating influence of a reflex action. Some of the observations and experiments in hypnotism, as those of Heidenhain[2] in particular, also throw some light upon the manner in which reflex epilepsies may develop into true organic cortical disease. The phenomena of unilateral hypnosis are particularly interesting in this connection. When certain definite cutaneous surfaces are irritated, certain muscles and groups of muscles related to these areas can be brought into isolated or successive action; stroking the ball of the thumb, for example, causes adduction of the thumb towards the palm; or stimulating the skin over the sterno-mastoid causes the head to assume the wry-neck position.

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[1] *Jour. Nervous and Ment. Dis.*, vol. xiv., No. 8, August, 1887, p. 478.

[2] '*Thèse pour le Doctorat en Médecine,*' 1878, Versailles, and *Rev. de Méd.*, Sept., 1885.

[3] *Rev. de Méd.*, Nov., 1885.

[4] *Arch. Gen. de Méd.*, Paris, 1887, ii., 5-19.

[5] *Lancet*, Lond., 1886, ii., 1211-1213, Abstract of Brown's Lectures.

A case reported by me in 1880,[3] is interesting in connection with this question. It was one of epilepsy clearly Jacksonian in type, and as clearly due to a fibroma involving a nerve trunk on the palmar surface of the hand. The patient, fifteen years old, had had the seizures since the age of four years, they coming on after an injury to the hand at the situation of the fibroma. After removal of the growth, she had spasmodic attacks of the same type as before the operation, but less in frequency for a year, after which she rapidly improved, and I have been recently informed has had no spasms for nearly two years.

A description of the usual character of the attacks shows that they were distinctly Jacksonian. The description will be quoted at length because of the importance of the matter under discussion.

The distal phalanx of the ring finger of the right hand was first flexed; secondly, a few spasmodic movements of flexion would occur in this finger; thirdly, the other fingers and thumb of this hand would begin to twitch convulsively—the second phalanges would be flexed, the last extended; fourthly, the clonic limbs as well as his head and trunk taking part in the spasm which was tetanic in character. In another case in which an operation was performed for me by Dr. Hearn, the spasm seemed to show a somewhat confusing admixture of what might be termed dural and cortical characteristics. Notes on this case were furnished to Dr. J. B. Roberts, and were published by him in his pamphlet on the 'Operative Surgery of the Human Brain.' In a fourth case seen with Dr. L. W. Steinbach, the patient had been subject to convulsions which seemed to date back to an injury to the head; he had a scar and apparently a depression of the skull over the frontal region. Pressure on this scar brought on a unilateral, largely tetanic convulsion on the same side as the scar. A flap including the scar was lifted, and trephining was performed, but nothing abnormal was found in the inner table or in the dura. The scar was excised. In a fifth case, the patient had convulsions, sometimes on one side, sometimes on both, and these could be brought on by pressure on a scar left by an old sabre cut. The cicatrix was cut out and the patient recovered; at least, he remained for several months in the hospital without attacks, although before the operation he had been having them at frequent intervals.

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[1] "Psychiatry, a Clinical Treatise on Diseases of the Fore-Brain." Translated by B. Sachs, M.D.

[2] "Hypnotism or Animal Magnetism." Translated by L. C. Woolridge, M.D.

[3] *Phila. Med. Times*, December 18, 1880.

With Franck I fear we are not always able to make a trenchant separation between cortical and reflex epilepsies, but a few points may be indicated. In reflex epilepsy the attack does not begin with brusque tetanisation as in the case of cortical disease. If the reflex epilepsy has a tonic period it rises slowly to its maximum. In cortical epilepsy the convulsio begins without exception on the side of the body opposite to the side of the brain excited or irritated; in reflex epilepsy, or at least in dural and perhaps other forms of trigeminal spasm, it frequently begins on the same side as the focus of irritation. Unfortunately we have not here a radical difference, as it may begin on either side in reflex cases. In the reflex cases, if the spasm begins locally or unilaterally, there is not likely to be a definite initial or signal symptom and serial order of movement; one half of the body usually plunges immediately into spasm.

True Jacksonian epilepsies are, I believe, sometimes reflex in origin; that is, they become established as the result of intense persistent peripheral irritation, dural, dental, palmar, &c.; and even after the source of irritation is removed the cortical discharges continue. Herein perhaps lies the explanation of Jacksonian spasm in which gross lesion is not discovered, "The animal had nine grand attacks in succession in twenty-five minutes. Then exhaustion came on and it was quiet, the respiration rapid, the heart beats quick, and the arterial pressure much diminished.

"It was left to repose for half an hour, then wishing to apply a manometer to the femoral artery, an incision was made in the skin of the thigh. At this moment a new convulsive explosion came on and without pauses, in the same register as had been made in the former attacks." * * *

The nerves of the dura mater spring from the fifth pair, and are distributed nearer to the internal than to the external surface of the membrane, which explains why some lesions of the dura are more likely than others to lead to spasm. The difference depends, in part at least on the site and intensity of the lesion with reference to the internal and external aspects of the membrane. Sub-dural hæmorrhage is more likely to give rise to reflex spasms than extravasation between the membranes and the skull, unless the blood tears through the membrane. A spicule of bone, in like manner, driven into the dura is more likely to cause reflex dural spasms than a depressed fragment; while a tumour arising in the membrane is more likely to bring about the same result than an exostosis, or a neoplasm growing from the agglutinated membranes into the brain substance, as is so often seen in intra-cranial growths.

I have notes of five cases in which operations have been performed for epilepsies apparently reflex in character. In two of these spicules of bone were removed from the dura mater. These cases bear out to some extent the views of the existence of distinctive characteristics for reflex epilepsies, but also point to certain resemblances to cases of cortical epilepsy. In one case in which fracture was present in the left frontal region, anterior to the motor area, the patient had convulsions at irregular intervals of weeks or months, usually having sharp pain at the seat of the scar before the seizure. He had no loss of sensation nor paralysis. His convulsions were frequently unilateral; I saw him in one which was confined entirely to the left side, and began in the left leg. This case was trephined for me by Dr. W. J. Hearn, of Philadelphia, and a spicule of bone dissected from the dura mater. In another case the patient was trephined by Dr. J. W. White at my request, for a fracture from a pistol-shot wound just above the right temple. Nearly three months after the injury he began to have spasms; and had had about seven seizures in all. In a convulsion which I witnessed he was completely unconscious; his body was twisted somewhat to the right; his face and all his one or several parts of the body, the movements of the limbs on the same side being more energetic than those on the opposite side. Franck has made careful comparison and contrast of cortical epilepsies and those which are reflex and toxic, including those which are due to irritative lesions of the dura mater. In one of his experiments[1] he produced an epileptic seizure from mechanical irritation of the dura, and among other things noted was that at the moment of the irritation of the dura mater the muscles of the face of the same side were attacked with violent convulsions. Attacks followed, as many as nine in twenty-five minutes, all clonic and generalised.

The following is an abstract of the record of this experiment:

"Experiment No. 45 (Jan. 7, 1879) with M. Senna of Coimbra. Reflex attack of epilepsy (excitation of the dura mater) commencing on the side irritated.—State of disease.—Arrest of the salivary flow in the attacks.—New series of reflex attacks by incision in the skin.—Circulatory modifications in this form of epilepsy.

"A young dog, spaniel of large size, very vigorous. The motor zone was exposed at the right side while under the influence of a slight anæsthetic of chloroform; a large opening was made in the frontal sinus in order to discover the excitable region, the crucial edge forward.

"It was proposed to study at the same time with salivation, the modifications of the heart and of compression in their connection with the cortical origin of convulsions; but the animal was taken with a reflex attack of epilepsy under the following conditions: The dura mater had been cut all round the trephining point; a fragment remained adherent to the anterior inferior angle of the wound and caused a slight flow of blood somewhat interfering with the experiments at excitation. At the time when an attempt was made to stop the flow of blood with a piece of medicated cotton, the simple friction of the strip of dura mater provoked a series of violent convulsive attacks, having their point of commencement in the muscles of the face and neck on the *same side* (contrary to the epileptic attacks of cortical origin, which *always* commence in the opposite side of the body, and severely in the corresponding muscles to the cortical centre excited).

"The first attack was exclusively clonic, very violent and generalised.

"During several minutes spontaneous attacks succeeded, separated from one another by a few seconds only; at the fourth attack the convulsive movements and the salivary escape were simultaneous. It was remarked in the first periods of the attack the salivation came on slowly; it appeared in this fourth attack only thirty-two seconds after the commencement of the clonic movements.

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[1] *Op. Cit.*, p. 470.

of whatever local origin, may cause unilateral convulsions or even monospasm. Brown-Sequard has contributed largely to our knowledge of this subject both in his early and recent researches. During a few years stimulated by practical specialism, much work in the direction of diagnosis and treatment of reflex epilepsy has been done, some of the best of it by members of the Associations represented in this Congress. The same conclusion might be arrived at for all, as that of Boucheron[1] with reference to aural epilepsy, namely, that spasm may proceed from lesions of the ear, eye, nose, pharynx, larynx, face, scalp, or dura mater, and may present all the clinical varieties of epilepsy, or even a form of hystero-epilepsy; and that the point of origin of these disorders is intense excitation of a sensory nerve.

Dural epilepsies are especially worthy of attention. During a recent operation in one of the Philadelphia hospitals a faradic current accidentally applied to the dura mater almost instantly produced spasm which invaded the whole body. Dupuy[2] has published various papers regarding irritation of the dura mater causing muscular movements, claiming that his results are constant when the animal experimented upon is not in a state of anæsthesia incompatible with the manifestations of animal life, and when it has not lost too much blood. Brown-Sequard and Burdon Sanderson have recorded similar phenomena; and the facts of these experiments have been used as arguments against cortical localisations. The Committee of the New York Society of Neurology and Electrology, in 1874 found that galvanisation of the dura or other sensitive parts produced by reflex action muscular twitchings, oftenest on the same side of the body. Duret[3] has given particular attention to the rôle played by the dura mater in the production of sensory, spasmodic, and other phenomena. He does not however, with Dupuy, hold that the fact of the production of spasms in this way in the least invalidates the doctrine of cortical motor localisation, but that a clear differentiation between dural and cortical spasm can be made. Rochefontaine has shown that irritation of the dura mater determined cries of pain and general movements more or less energetic; and also that mechanical irritation of the sensitive points of the membrane produced in certain conditions movements limited to

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[1] *Compt. rend. Acad. de Sc. Paris*, 1887, cv., 944-947.

[2] *Examen de quelque points de la physiologie du cerveau.* (Thèse inaugurale, Paris, 1873.) Also: *Experiences sur les functions motrices du cerveau*, 1888. *Compt. rend. des séances de l'Acad. des Sci.*

[3] '*Sur les Traumatismes Cérébraux*' and *Brain*, April, 1878.

CORTEX : HEARING.

27. The first temporal convolutions right and left in a person deaf with the left ear. MANOUVRIER (*Revue Philosoph.* xiv., 3, p. 330).

CORTEX : SALIVARY SECRETION.

28. Secretion of Saliva on stimulating the Cortex. ECKHARD (*Neurologisches Centralbl. Feb.* 1, 1889).
29. The question of the Cortex areas which induce secretion of saliva. BECHTEREW and MISLAWSKI (*Neurolog. Centralbl., April* 1, 1889).

CORTEX : TEMPERATURE.

30. Thermo-electric observations upon the Cerebral Cortex in various emotional states. TANZI (*Revista Speriment. di Freniatr.* xiv. p. 234, 1888).

Variations of 1°-2° and in some instances 3° C. observed in the dog, independently of alterations in pulse and respiration.

CORTEX : DEGENERATION.

31. Changes in the Cerebral Cortex in a case of blindness and deafness developed in early childhood. TOMASCHEWSKI (*Centralbl. f. Nervenheilkunde* xii. v. 1, p. 21, 1889).

CORTEX : ELECTRIC CONDUCTIVITY.

32. Induced unipolar currents in studying the electrical excitability of the brain. NEGRO (*Arch. ital de Biol.* xi. ii. pp. 212-225, *March,* 1889).

CORPUS CALLOSUM.

33. A case of absence of Corpus Callosum in the human brain. BRUCE (*Reports from the laboratories of the Royal Coll of Physicians, Edinbro'*, 1889. *Medical Record* '5, p. 87).

BODY.

34. Multiple of the blind-worm. DUVAL and KALT , 1889, *No.* *Biologie* 1889, p. 85, *Feb.* 9).

PINEA)PHYSIS.

35. Certain s pineal eyes development of the Hypophysis Cerebri (*C. R. Soc. de* *rh. d. B in Stockholm I.* 3, *Dec.*, 1

HYP

36. Anatomica tages in the ophy of the left Corpus 88). LECHE (*V* TIMMER (*Nederl. Tijdschr.*

CORPUS MAMMILLARE.

Remarks on a Case of Atr ammillare. WINKLER voor *Geneesk.* 26

Left hemisphere atrophied in its convolutions, but not much so on the base. Left corpus mammillare atrophied. Fimbria in front of descending horn well developed, but very thin where it began to curve. Pulvinar and anterior tubercle of thalamus were atrophied, and the thalamus shorter than normal. No distinct atrophy of the optic nerve or its connections. The cortex of the gyrus hippocampi and subiculum cornu Ammonis were slightly atrophied, the left crus fornicis and columna ascendens were strongly so. Observations upon the course of the columna ascendens of the right fornix.

PYRAMIDAL TRACTS.

37. The Pyramidal Tracts in certain Mammals. LENHOSSÉK (*Anat. Anzeiger, April* 20, 1889, pp. 208-219; *woodcuts*).

Mouse, guinea pig, rabbit, cat; the pyramidal tracts much less developed than in man, as estimated by relative cross-sections at birth. They also occupy different situations in different animals. In the mouse and rat they lie in the posterior columns, in the rabbit and cat in the lateral columns, in man partly also in the anterior columns. In all animals observed the tracts completely cross.

38. The Intercrossing of the conducting paths serving voluntary movements. BROWN-SÉQUARD (*Archives de Physiologie*, 1889, 1 and 2, pp. 219-246).

39. Nerve-tracts degenerating secondarily to lesions of the Cortex Cerebri. SHERRINGTON (*Journal of Physiology*, x. 5, pp. 429-432; *July*, 1889).

Preliminary account of the results of observations made with a view of determining the exact number of fibres degenerating in the pyramidal tracts after remov s portions of the cerebral cortex, their situation ion in the cord. S. finds that when arm centre onl degenerated fibres are found even throughout the sa f the cord. After injury to leg centre many de val of variou es stop short at cervical swelling. Degenerated and extens nd in both lateral tracts—those on the same side a is injured being fibres which have "re-crossed", eighbo ral regions e cervical and lumbar enlargements. nerated fib erated fibres may increase from abo bres are fo e to division of fibres. Two fibres lying the lesion l fibres" being found in the crossed in the ne urhood of t very much less frequently in th The number of dege not definitely grouped

ve downwards. This is d
close together or "gemina
pyramidal tract, and
Fibres are n

in the tracts according to the cortical area from which they come.

The fornix-fibres degenerate from behind forwards. Cortical lesion produces degeneration in the corpus callosum, the fibres do not unite identical areas but tend to scatter.

40. Appendix to papers on descending degeneration following lesions in the Gyrus Marginalis and Gyrus Fornicatus in Monkeys. FRANCE (*Proc. Roy. Soc.*, 1889, p. 122).

Tends to establish a differentiation in the pyramidal tract.

CEREBELLUM: HISTOGENY.

41. Histogenesis of Cerebellar Cortex. BELLONCI and STEFANI (*Archives ital. de Biologie*, XI., p. 21, *Jan.* 1889).

Deals with origin of molecular layer and relation to its growth of the external nuclear layer of embryonic brains. The latter is regarded as a germinal layer similar to the malpighian layer of the skin. From its cells originate the radial fibres of the molecular layer.

CEREBELLUM.

42. Atrophy of Fibres in the Cortex Cerebelli. MEYER (*Archiv. f. Psychiatrie*, xxi. i., pp. 197-222, pl. VII., *August*, 1889).

Observation of the medullated fibres in the cortex cerebelli in progressive paralysis and similar diseases.

The cerebellum suffers in the same kind of way as the cerebrum, especially its frontal lobes, and on the same side. There was but little alteration in the number or appearance of Purkinje's cells.

MEDULLA.

43. Nuclei Arciformes. JELGERSMA (*Centralbt. f. Nervenheilkunde*, No. 7, 1889).

These nuclei atrophy in the same way as the nuclei pontis. Like the nuclei pontis they are undeveloped or badly developed in many idiot brains. T. supposes that the fibres which originate in these nuclei are connected on the one side with the cerebellum through the restiform body, on the other side with the great brain by fibres lying in the pyramidal group. T. regards the nuclei as not belonging to the arciform fibres, but as pontal nuclei which have been displaced caudalwards.

CENTRES: LARYNX.

44. Motor-centres of the Larynx. MASINI (*Archivio ital. de Laryngologia*, viii., p. 45, 1889).

NERVES: CRANIAL. I.

45. The Nose and Jacobson's Organ. BEARD (*Morphological Studies*, iv. *Jena, Gustav Fischer*, 1889. *Reprint from Zoologische Jahrbücher*).

Contains an account of the development and structure of Jacobson's organ in the Ophidia and Lacertilia. Concludes that the organ is a specialised part of the nose of much greater sensitiveness than the rest. Larger part of paper devoted to a consideration of the segmental meaning of nose. B. is strengthened in his conviction that the nose and the ear, quite unlike the eye, belong to the same category of sense organs as those of the lateral line or branchial sense organs. Bibliography of Jacobson's organ.

OPTIC CHIASM.

46. The Development and Course of Medullated fibres in the Optic Chiasm of Man. BERNHEIMER (*Archiv. f. Augenheilkunde*, xx., 1 and 2, pp. 133-179, pl. x. *June*, 1889).

NERVES: CRANIAL. II.

47. Experimental and Pathological Observations upon the Optic Centres and Tracts. MONAKOW (*Archiv. f. Psyschiatrie*, xx., 3, pp. 714-788, pts. xi., xii., xiii., 1889).

Anatomical study of brain of cats and dogs operated upon by M. and Munk.

NERVES: CRANIAL. II.

48. A New Centre for the Optic Nerve in the Hen. PERLIA (*Archiv. f. Ophthalmologie*, xxxv., 1, pp. 20-24, pl. ii., *April*, 1889).

By Gudden's method a portion of the optic tract is followed in the hen beyond its usual end-station in the mid-brain to a nucleus (the nucleus of the median optic tract) in the lateral part of the roof of the after-brain on the outer side of the trochlear nucleus.

NERVES: CRANIAL. II.

49. Optic-conducting Paths in Human Brain. RICHTER (*Archiv. f. Psychiatrie*, xx., 2, p. 504).

Atrophy of the optic nerves on one or both sides can, when it affects a grown-up person, be followed with the naked eye as far as the pulvinar and external geniculate body, but after it has existed for a year it will not necessarily be seen on macroscopic observation.

Cranial Nerves. III.

50. The Upper Nucleus of the Oculomotorius. Darkschewitsch (*Arch. f. Anat. u. Phys. Anat. abth.*, 1889, i. and ii., pp. 107-117, pl. vii.).

In the *Neurologisches Centralblatt* Darkschewitsch has described, in the human fœtus, a collection of cells just above the oculomotor nucleus. This he termed the "upper" or "small celled" oculomotor nucleus. He finds that this nucleus is in connection with the ventral fibres of the posterior commissure, which are the first to myelinate.

The upper nucleus has, at least, three connections; (1) with the posterior longitudinal bundle; (2) directly with the pineal gland through the ventral part of the posterior commissure; (3) by certain fibres originating in the nucleus it takes part in the formation of the ansa lenticularis. The posterior commissure contains at least two sets of fibres, neither of which belong to the formatio reticularis.

Cranial Nerves. VIII.

51. Origin of Auditory Nerve in the Rabbit. Bumm (*Allg. Zeitschr. f. Psychiatrie*, xlv., 5 and 6, p. 568).

Cranial Nerves. VIII.

52. Origin and Central Course of the Nervus acusticus in the Rabbit and Cat. Baginsky (*Sitz. der K. P. Akad zu Berlin*, xxxiii., p. 635, *June* 27, 1889).

Destruction of the labyrinth in young animals and examination of the brain seven to eight weeks after the operation by Weigert's staining. Cochlea destroyed on right side, atrophy of posterior root, anterior auditory nucleus and tuberculum laterale. Also on this side considerable shrinking of corpus trapezoides and upper olive, while the upper olive of the opposite side was normal. Deiter's nucleus, inner auditory nucleus, corpus restiforme, pons, cerebellum, brachium conjunctivum, posterior longitudinal bundle unchanged. Atrophy of opposite inferior fillet as far as posterior brachium, but no obvious atrophy of posterior quadrigeminal tubercle could be observed. Extensive atrophy of the striae medullares of the same side. Great difficulty in causing atrophy of the anterior root of the auditory nerve by destroying all the ear except the labyrinth. It was accomplished in two rabbits and one kitten. On the same side the inner division of the cerebellar peduncle (*Meynert*) was diminished in size and its scattered cells atrophied. Small nerve cells on the ventral side of Deiter's nucleus were atrophic. Deiter's nucleus not considered to be in connection with the auditory nerve.

CRANIAL NERVES. X.

53. Minute Anatomy of the Vagus Nerve in Selachians, with Remarks on the Segmental Value of the Cranial Nerves. SHORE (*Jour. of Anat. and Phys., April,* 1889, pp. 428-452, pl. xx. and xxi).

Critical account of previous work on the subject. Method—osmic staining. Conclusions: (1) The vagus in the skate presents a small group of cells on the hindermost of the posterior roots equivalent to dorsal root ganglion. A group on each branchial branch—vagrant ganglia of splanchnic motor roots. A second group on each branchial branch—the pre-branchial ganglia. A group in the visceralis. A group in the lateralis. (2) The vagus of Raia does not contain any non-ganglionated somatic motor fibres. It is not, therefore, a complete metameric nerve (4) It contains the typical element of the so-called sympathetic system; the proximal ganglia are represented by the branchial and visceralis ganglia, the distal by the pre-branchial ganglia. As will be seen from the extract, *Gaskell s* views and nomenclature are adopted.

CRANIAL NERVES. XII.

54. A rudimentary Dorsal Root with a Ganglion for the Hypoglossal Nerve in Man. CHIARUGI (*Boll. R. Accad. dei Fisiocritici di Siena,* vi., ii.; *Arch. ital. de Biologie* xi., iii.).

Small filament with ganglion accompanying the most caudal roots of the hypoglossal in a child of two. The ganglion appeared to belong to filaments of the dorsal roots of the first and second spinal nerves.

CORD—TOPOGRAPHY.

55. Relations between the Superficial Origins of the Spinal Nerves from the Spinal Cord, and the Spinous Processes of the Vertebræ. REID (*Jour. Anat. and Phys., April,* 1889, pp. 341-354, pl. xiii., xiv.).

After describing method adopted for obtaining an orthographic projection, Reid gives results for each root in his specimens. Finding that "the superficial origin of any individual spinal nerve has no fixed and definite relation to the apex of one, or the apices of two spinous processes, or the space intervening between two, but varies considerably," the author gives a table showing highest and lowest point at which root was situated in the specimens examined.

mentary changes observed in the fresh state. Splanchnic and other ganglia also observed. Conclusions that (1) in lower mammals and young human beings the collateral ganglia are functionally active, but that in monkeys there are evidences of the commencing loss of this function, which has completely disappeared in the human adult. (2) In man the function of the lateral ganglia is maintained well into adult life, and only begins to disappear in old age.

65. The Effects of Extirpating the Cæliac Plexus. LUSTIG (*Archivio per le Scienze Mediche*, xiii. p. 129, 1889).

This experiment has yielded various results in the hands of different physiologists, such as diabetes insipus, diabetes mellitus, alterations of digestion, atrophy of the pancreas, &c. Some of these effects may be due to peritonitis. *Lustig* reduces this to a minimum by antiseptic precautions. Rabbits and dogs die, as a rule, in two to three weeks suddenly without alarming symptoms. For first day or two sugar usually appeared in the urine, but subsequently disappeared. Then acetone appeared, and later albumin; the urine diminished in amount; hyaline casts, red and white blood corpuscles, and epithelium were present. In two rabbits which survived acetonuria and albuminuria disappeared.

SYMPATHETIC—VASO-MOTOR.

66. Innervation of the Renal Blood Vessels. BRADFORD (*Journal of Physiology*, x., 5, pp. 358-408, pls. xxiii.-xxvi., *July*, 1889).

67. Method for completely Destroying the Cord in Mammals. Application to the analytical study of Vaso-motor Actions. GLEY (*Comptes Rendus de la Soc. de Biol.*, p. 110, *Feb.* 16, 1889).

In a dog under artificial respiration the medulla is cut across between the skull and the atlas. An opening is made into the spinal canal in the lumbar region. A catheter is passed into the upper opening; and the cord washed out of the lower opening in fragments in from twenty to twenty-five minutes without the least bleeding. The influence of local agencies upon the vasomotor system was then studied.

68. Vaso-motor nerves of the Head. MORAT (*Archives de Physiologie*, 1889, 1 and 2, pp. 196-211).

NERVES—VISCERAL.

69. Centres for the Innervation of the Small Intestines. PAL and BERGGRUN (*Mediz. Jahrbucher*, viii., 1888, [*issued June*, 1889]).

Centres for inhibition of the small intestine must lie above the thalamus, the fibres passing through the medulla and corpus striatum to the sigmoid gyrus.

SYMPATHETIC—REFLEX.

70. The Presence of Reflex Vasomotor Centres in the Ganglia of the Sympathetic System. ROSCHANSKY (*Centralbl. f. d. Wissensch. Med.* 1889, p. 162).

The cutting of the cervical cord in curarised cats does not completely prevent the elevation of temperature which is produced by stimulating the central stump of a splanchnic nerve, although it diminishes it. Nor is it altogether abolished by destroying the spinal cord below the level of section, the animal being kept alive by transfusion of defibrinated cat's blood. It is not abolished by cutting the lateral chain between ninth and tenth ganglia, but the pressure is greatly lowered by dividing it between the origins of the great and lesser splanchnics.

NERVES—PERIPHERAL.

71. Innervation of the Muscles of the Soft Palate. TURNER (*Jour. Anat. and Phys.* 1889, *July*, pp. 523-532).

NERVES—DISTRIBUTION.

72. The Localization of Skin Sensibility in its relation to the Sensory Roots of the Spinal Cord. BOCCI (*Moleschott's Unters. z. Naturl.*, xiv. 1, p. 19).

In the frog the posterior root of the seventh lumbar nerve supplies the skin covering the triceps femoris, that of the eighth nerve the skin over the peroneus and tibialis, that of the ninth the skin over the gastrocnemius.

ELECTRIC ORGAN.

73. Electrical Organ of Skate. SANDERSON and GOTCH (*Journal of Physiology*, x. 4, pp. 259-277, pl. xxii. *May*, 1889).

CONSCIOUSNESS.

74. Consciousness and its Limits. BECHTEREW (*Kasan*, 1888, in Russian, *Abstr. Neurologisches Centralbl.*; *May* 1, 1889).

PERCEPTION.

75. The Production of a perception of Pain by Summation of Sensory Stimuli. NAUNYN (*Arch. f. exper. Path. und Pharmak.* xxv., iii. and iv. p. 272, 1889).

INHIBITION.

76. Field of Action of Inhibition in Physiology, in Pathogeny and in Therapeutics. BROWN-SÉQUARD (*Archives de Physiologie*, 1889, 1 and 2, pp. 1-24).

REFLEX—KNEE.

77. On the Nature of the Knee-jerk. LOMBARD (*Journal of Physiology*, x., 1 and 2, pp. 122-149, *February*, 1889).

CELLS.

78. Nerve Cells as Centres of Energy. BOCCI (*Moleschott's Unters. z. Naturl.* xiv., 1, p. 16).

HISTOLOGY.

79. Is the Active Condition of the Central Nervous System accompanied by Changes visible with Microscope? KORYBUTT-DASZKIEWICZ (*Arch. f. Mikros. Anat.*, xiii., 1, pp. 51-71, *June* 12, 1889).

Two exactly similar frogs chosen. Sciatic plexus of one stimulated for one hour. Spinal cords of the two frogs then prepared in exactly the same way (hardened in corrosive sublimate, &c.). Sections of each cord stained in hæmatoxylin, nigrosin, eosin, and safranin. The nuclei (? of nerve cells) stained some blue, others red. In the stimulated animal the proportion of red nuclei to blue nuclei was much higher than in the control animal.

80. Certain Constituents of Peripheral Medullated Nerve Fibres. JOSEPH (*Sitzungsb. d. K. P. Akad zu Berlin Dec.* 18, 1888).

Hardening in ½% osmic acid two hours. In the electric nerves of the torpedo, the axis cylinder is to the myelin sheath in cross section as 3-5. In Lophius, frog and rabbit the relation was the same. Two different substances differentiated both in the medullary sheath and in the space in which the axis cylinder lies, that in the former being indentical with *Ewald* and *Kühne's* neurokeratin. The axis cylinder scaffolding remains intact after the nerve has been cut for fourteen days and is therefore considered to be non-nervous. Joseph concludes that the axis cylinder contains two substances (in addition to the scaffolding).

81. Commencement of Schwann's Sheath in the Spinal Roots. SPRONCK (*Nederl. Tijdschrift voor Geneesk*, p. 147, 1888).

The first nodes of Ranvier which appear in the roots being placed all at about the same level form a characteristic flat group.

They lie either within or without the cord and make a morphological boundary between the naked cylinders and the peripheral fibres invested with Schwann's sheath. The commencement of the sheath is conical and it is here surrounded by neuroglea cells which can be followed some distance into the root. The first internode is as long as the succeeding ones, but in a few segments the want of regularity has scattered the nodes, so that they are no longer grouped in a plate.

HISTOGENY.

82. The Development of Certain Cerebral and Spinal Nerves. CHIARUGI (*Anat. Anzeiger*, pp. 31-32, *Jan.* 10, 1889).

83. The Development of the White Matter and Extension of Fibres in the Spinal Cord of the Mouse. LENHOSSÉK (*Arch. f. Mikros. Anat.*, 1889, i. pp. 71-125, pls. 6, 7).

A repetition upon the mouse of Flechsig's work upon man. Conclusion that the appearance of the myelin sheath in the central nervous organs of vertebrates, and especially higher vertebrates, follows the same laws as in man.

(1) Gelatinous substance of Rolando reaches to the periphery; it consists of cuticularised (verhornten) epidermal cells without distinction into connective tissue and nervous elements. (2) Nerve cells and fibre network. (3) White substance and calibre of fibres. (4) Anterior roots. (5) Anterior commissure. (6) Anterior columns. (7) Lateral columns. (8) Posterior roots. (9, 10, 11) Posterior columns and posterior commissure. (12) Pyramidal tracts, successively treated in detail.

84. The Neuroblasts and their appearance in the Embryonic Spinal Cord. HIS (*Archiv. f. Anat. u. Phys.*, 1889, iii. and iv., pp. 249-300, pls. XVI.-XIX.)

A continuation of the investigations into the origin of the differentiation of the several elements of which the cerebro-spinal axis is composed. The medullary plate consists at first of a single layer of cells. Between the bases of the epithelial cells, protoplasmic or germinal cells are seen. The epithelial cells are changed into "spongioblasts," which give rise to a network or "scaffolding." The interior of the cells exhibits a distinction into a formed filamentous substance and a transparent soft substance. The former enters into the formation of the network which unites neighbouring cells. On the interior of the axis it constitutes the inner limiting membrane; on the outside it collects into a thick, spongy plate, the "velum confine" (if a synonym

CIRCULATION—SINUSES.

87. Experimental Obliteration of the Sinuses of the Dura Mater. FERRARI (*Arch. ital. de Biologie* xi., 1, p. 171, *Jan.* 1889).

CIRCULATION—LYMPH.

88. Cerebro-spinal Fluid. HALLIBURTON (*Journal of Physiology* x., 4, pp. 233-253, *May*, 1889).

CIRCULATION.

89. The Cerebral Circulation in Man in the Normal State and under the Influence of Hypnotic Substances. RUMMO and FERRANNINI (*Resumé in Arch. ital. de Biologie* xi., 2, pp. 272-338, *March*, 1889. 4to, pp. 69, 16 *lithographs* and 17 *engravings, Terani, Naples*).

Advantage taken of cases of deficiency of the skull. Movements of brain transmitted to a tambour. Variations due to time and meals. During sleep three phases distinguished—(1) From ten to one a.m. ischaemia with constriction of vessels and diminished frequence of pulse; (2) one to three, slower pulse dilated vessels; (3) three to five, increasing vaso-constriction with more frequent pulse. Plethysmographic observations of variations in circulation in limbs, accompanying the brain changes. Similar observations under chloral, paraldehyde, morphine, narceine, hypnone, methylal, and urethane. Some substances, such as narceine and morphine, modify rather the excitability of the nerve centres; others, such as hydrate of choral, paraldehyde and ethyl urethane the cerebral circulation; others, hypnone and methylal, for example, modify the one and the other almost like physiological sleep.

CORD—NUT

90. Experiments upon the r
Cells of the Medulla
SARS (*Virchow's Arc*

N

91. Congenital Neurop
tioner

Quoted in this place
of nutrition. Girl æ

looking papules, itching intensely. One patch corresponded to external branch of radial, the other to cutaneous branch of ilio-inguinal. The first cured by dividing the nerve, the second treated with potassa fusa recurred.

NERVES : IRRITABILITY.

92. Action of heat on muscle and nerve fibres. MORIGGIA (*Archives ital. de Biol.* xi., 3, pp. 379-389, *June*, 1889).

Frogs plunged in baths of hot water or oil. Nerves retain their irritability at temperatures at which muscles coagulate.

HEAT.

93. Thermopolypnoeic centre and thermotaxis. OTT (*Phil. Med. News*, 1889, *No.* 4, p. 109).

MICROCEPHALUS.

94. Description of three microcephalus brains, and anatomical study of microcephalus. Part I. MARCHAND (*Nova Acta der k. Leop-Carol. Akad. d. Naturforscher*, Bd. L. iii., iv. 3, 5 plates, 1889).

MICROCEPHALUS.

95. Encephalon and Skull of a Microcephalus. MINGAZZINI and FERRARESI (*Moleschott's Untersuchungen z. Natur. d. Menschen u. d. Thiere* xiv., 1, p. 103).

METHOD.

A Modifi on of Weigert's Method for coloring the nerve
centre ASSALE (*Riv. speriment. di Freniatria, &c.,*
88

fluid or bichromate of potassium.
by Weigert.
t distilled water 100 gr.
neutral copper acetate.
cyanide 2·5 gr. in 300 gr.
and an equal time in B.
us decolored, dehydrated in
olic acid to 3 parts xylol

Wales, Mr. Parnell," and other celebrities, and values the same at £1,000 apiece. "Possesses an instrument the size of this house for taking photographs; a room full of gold watches to give away," &c. Denies that he is any exalted personage, however, but is only plain "P. K." "Some years ago drank very heavily, and has been in poor circumstances of late, but never without a gold lever watch, worth £20. Is as good a man as ever he was." Gives a pretty complete history of constitutional syphilis. Confirms the absence of any neuroses in his family, and says that his grandmother is in Leeds Workhouse, 108 years of age, and two feet high.

Physical condition on admission.—Head rather square-shaped, facial expression beaming or else lachrymose, frontal lines fairly marked, naso-labial fold almost obliterated; skin of face, in part with that of body, sallow and greasy. Irides bluish, free from discolouration; pupils accurately circular, unequal in size, right the larger; do not react to light, act with accommodation. Tongue protruded straight, is tremulous and furred; speech rather laborious, and motions of lips clumsy and exaggerated. Patellar reflexes are absent, superficial brisk, no clonos. Emaciated and of feeble muscular development. Height 5ft. 3in.; weight 126lbs. Gait of ataxic kind; patient can stand fairly well with feet approximated and eyes closed, but is unable to balance himself on one foot; he states that he has often fallen down of late. There are scars from an old papular eruption and brownish discolouration of the skin, especially of abdomen and thighs.

Progress of mental symptoms.—At first restless, noisy and tiresome, soon settled down into mild good-humoured excitement, and
with the a[illegible]e of dementia had alternating states of expansive-
ness, exci epression. Generally, optimistic, pertina-
cious ar emely loquacious, and much of a pest to
e could run and walk well when hardly
. During periods of depression was
lained at such times of much shoot-
occasionally of severe gastic pain
during times of elation acknow-
riction, and that he had fre-
eral weeks prior to death,
his inability to stand; for
, sometimes weeping; not
of himself. One week
perfectly apathetic

Summary of physical condition.—Right pupil always the larger, 5 mm., left pupil, 3.5 mm. Act with accommodation, and are fixed to light. Very faint consensual reflex in both eyes. No dilatation with sensory stimuli, no strabismus, no diplopia, no irregularity of pupillary circle, no discolouration, no adhesion. Perception of colours good; ocular movements unimpaired. Vision defective, can read nothing smaller than "double pica" at fourteen inches. Much labial tremor, lips twitching convulsively during speech. Tongue straight, tremulous; no hemiatrophy. Speech slow, hesitating, slurred; voice quavering. Gait typically ataxic;
feet planted about ~~twenty~~ inches apart; lifted up high during
walking and the heels brought down ~~heavily~~; eyes fixed on floor
during progression; neighbouring objects clutched at for support.
Without holding on to something is quite unable to maintain
equilibrium, and with closed eyes totters and would at once fall
unless kept up. Ha ds are unsteady but patie at could write his
name fairly. No lo r as rough tests
decide. No defect in smell faulty;
says "assafœtida is musk, succeeds
with snuff. Taste punces quassia
sweet, quinine not v e good ginger-
beer"; detects suga t apparently
impaired; tactile se s of muscular sense, as fa to be some-
what deficient from f hearing. Discrimination ably so at
soles of feet. Eithe splendid;" fails to recognise ssions or
from his mental con so indiscriminative; he pron stimuli.
Dynamometer $\frac{21}{24}$. ly bitter, adding "it would mak wasted
and flabby. Calf-m readily. Thermo-sensibility no
(inco-ordination so g sation found by æsthesiometer
life that locomotion knees downwards, and considera
throughout; superfic (through retardation of sense impr
Post-mortem a lition, is sluggish in responding t so
normal, excep Iuscles of lower extremities are m uch w
The sk asurements are—right, $10\frac{1}{8}$; lef t, 10 inches
ho eat during the last two months of patient's
was impossible). Patellar-refle xes absent
al brisk at first, slow afterward. No clonos,
Thoracic and abdominal viscera

teriorly are almost completely converted into fibrous-tissue. From this cause the nerve-bundles have a shrunken, compressed aspect and have very generally absorbed the aniline-dye. Only a few unchanged nerve-tubules are seen. The vessel-orifices are both large and extremely numerous. The nerve-funiculi anterior to the cord seem quite healthy; the extreme end of the cord where the structure is mainly or entirely of grey matter, the same. Weigert-stained specimens corroborate all preceding description. In these last, too, the purple band of nerve-fibres sweeping across from posterior nerve-root to grey cornu is represented only by a few non-diseased tubules against a ground of connective tissue; so also in the case of the internal radicular fibres, whilst the vertical medullated tubes placed in front of the substantia gelatinosa are very sparse.

Lumbar cord.—Sections were taken from four planes. There is pronounced degeneration of the whole posterior columns, excepting a small narrow oval area on each side of the posterior third of median septum, and that immediately bordering the posterior commissure. Elsewhere the appearances are—diffuse aniline-staining from increase of connective and vessel-orifices, and its absorption by the medullated sheath of nerves. The outline of the great majority of nerve-fibres is quite indistinct from shrinkage and displacement. Some (the large) remain unaffected, others are cloudy and swollen, failing to show an axis-cylinder. A fine meshwork of dark dotted lines, formed by fine atrophied tubules and divided connective-fibrils traverses the field, whilst numerous patchy deep-stained areas of degenerate tubules are distributed. Towards the periphery the sclerosis is most intense; the post-commissural zones show also, with the fore-given exception, disease nearly as advanced as that in posterior columns generally. A considerable proportion of the morbid change is formed by the vessel-elements. The arterial tunics are much thickened, the thickness being equal, often, to the diameter of the lumen; are of homogeneous or vitreous aspect. The posterior halves of the columns are the richest in vessels, the most prominent being the septal, postero-internal, and its ramus passing into Goll's column. These are frequently bordered by compressed and deeply-stained nerve-fibres, and by dense fasciculi of connective-tissue, to which appearances are often added collections of large unstained granular cells, hereafter to be alluded to. The description of the entering posterior root-fibres, &c., given in the sacral region applies here. Those vertical tubules occupying an area corresponding to that of Clarke's column higher up, are extremely-few, and the medullated fibres of the posterior commissure plainly reduced in number.

Dorsal region.—In the lower dorsal segment the sclerosis in the posterior commissural zones is more advanced than in the lumbar cord, and extends quite up to the grey matter. This it does in the upper segment of this region also, and is most intense in Goll's column. In these zones, large tracts of connective-tissue, numerous vessel-orifices, areas of shrunken nerve-fibres and swollen, opaque tubules give a very patchy appearance. The latter, most plenteous—like the connective element—near the grey cornua, are found elsewhere in the posterior columns, forming by aggregation, dense-white, irregular patches, often mapped-out by a bordering of dark sclerosed nerve-fibres. In the columns posterior to the zones, a connective-tissue excess is not so marked, and the main constituents of the field are scattered nerve-fibres of fairly normal aspect, a broad marbling of partly degenerated tubules, fine tracts and areas of densely-packed, shrivelled fibres, capillary-orifices which actually riddle the section in parts, and collections of large pale granular cells, which occupy the perivascular spaces. In no other part of the cord (upper dorsal) are these latter bodies found in such number. They congregate around the median septum in company with abundance of leucocytes, and ramifying connective-cells, about the larger vessels, and elsewhere, in accumulations, often quite conceal the capillary, in the course of which they lie. The foregoing appearances, best observed at the periphery, are somewhat modified more anteriorly; the fine tracts of degenerate nerve-fibrils—running like irregularly-strung wires of a sieve—give place to patchy areas; by involvement of intervening neurine structure; so that approaching the anterior fourth of the columns the field appears to consist of a deeply-stained groundwork of nerve-fibres, closely packed, studded with the nearly black puncta representing the axes—the appearance occasionally resembling the transverse section of a wire-cable. With few exceptions there is little intermediate between these areas of intense degeneration and well-defined nerve-tubules of fairly-normal aspect. The large, cloudy, irregular opacities encountered, in which are occasionally seen indistinct, swollen, almost colourless granular axes, appear to be the sections of moniliform varicosities of tubules.

Cervical region.—In the lower portion, though still almost universal in the posterior columns, the sclerosis is not so dense next the grey matter, and the periphero-internal part of Burdach's zone is becoming approximately healthy; an excess of

ILLUSTRATIONS TO THE PAPER ON PATHOLOGICAL ANATOMY OF A CASE OF TABES DORSALIS WITH GENERAL PARALYSIS, BY MR. F. ST. JOHN BULLEN.

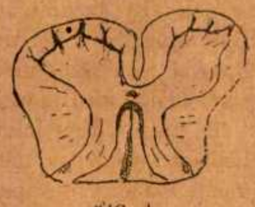


FIG. 1.

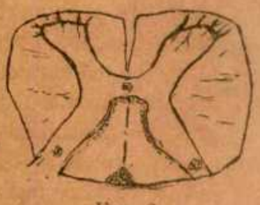


FIG. 2.

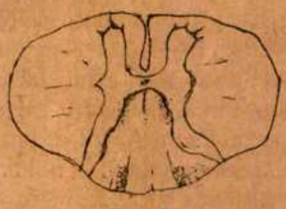


FIG. 3.

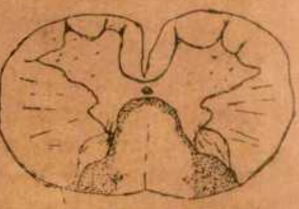


FIG. 4.

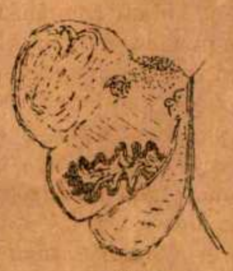


FIG. 5.

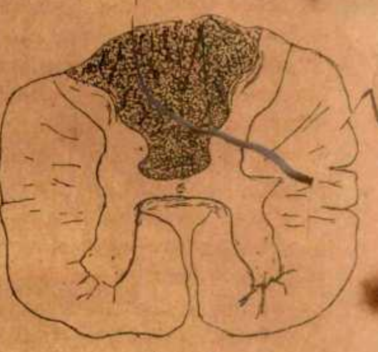


FIG. 6.

EXPLANATION OF FIGURES.

FIGS 1, 2, 3, 4.—Sections of Spinal Cord in the lower and upper lumbar, and upper dorsal and cervical regions. The dotted areas represent those occupied by healthy medullated tubules.

FIG. 5.—Medulla Oblongata.

Sites of lesion indicated by dotted areas; viz.:—floor of ventricle, vagal nucleus and funiculus solitarius.

FIG. 6.—Degenerate area in lower dorsal region.

could justly be compared; about a fourth of the remaining cells were of dubious character, the others, 137 in number, being highly atrophied or converted into pigment masses. Of the cells in the diseased cord, accepted as healthy, eighty-five existed on the right to sixty-eight on the left side. In the mid-dorsal region they are, in same relation, as twenty-five to eleven. Although thirty-four sections were taken from the upper dorsal segments the difficulty in counting and grouping cells, according to previous method, in these regions made the results uncertain. It may be reiterated that, notwithstanding due allowance made for the variations of cell-form, &c., always encountered even in presumably healthy cords, the appearances found denoted conclusively a diseased state of the columns. Where the latter are best developed, a great number of cells are converted into irregular masses of pigment without trace of cell-wall or nucleus, shrunken within pericellular space and surrounded by round nucleated-elements. The vertically-running fibres of the columns, instead of closely dotting their area, are almost absent, even in the anterior portion; those fibres passing between posterior root-zone and vesicular column and sweeping around and across it, are barely indicated; those obliquely placed, running outwards to processus lateralis and backwards to cornu posterius are well marked, but not traceable into the cell-area itself, there being as aforesaid but few in that situation. Just anterior to the vesicular column is a group of nerve-tubules of considerable proportions running upwards and outwards, in the latter direction traceable for a long distance into the lateral column. They appear intact. Many of the preceding features are well confirmed by vertically-made sections.

Medulla.—The following account is abbreviated from the notes relating to the above.

Decussation Region—Clavate and Cuneate Nuclei.—The grey nucleus of the median column is of badly-differentiated outline, the medullary neurine-structure in which lying plainly degenerate, and its elements huddled together, compressed and wasted, so that the field presents a disorderly, ill-arranged aspect; patchy areas of punctate-tissue, numerous capillary-orifices, connective-nuclei and amylaceous bodies are freely scattered. The cells are evidently atrophied, often pigmentarily degenerated and in great number, apolar. Those in the cuneate nucleus show quite as notable changes; the postero-external group, however, is an exception, the changes not being so marked there. Degeneration in the cuneate nucleus is much more unmistakeable in the section-planes including the lower convolute of the olivary body.

Substantia Gelatinosa—Has an ill-defined appearance owing to morbid change in the medullated fibres lying within the interstices of its branching grey-matter; these are pretty generally involved in wasting and are rather diffusely-stained by aniline. A considerable excess of capillaries also appears.

The arcuate bands in proximity to the central column are certainly both thinner and fewer. Those placed at periphery—stratum zonale—are unaffected, but the usually-seen broad bands traversing the zonal area of the fifth roots are not visible. The fasciculi proceeding from the raphé to and around the funiculus solitarius are (at the planes below the level of the vagal nuclei) distinctly thinned, and at times almost indistinguishable, or appear as few isolated fibres lying in a connective band.

In a healthy medulla, at and below the planes of the hypoglossal nucleus and before the opening up of the central canal, are seen obliquely-cut sections of numerous small bundles of nerve-fibrils, which lie at the postero-lateral part of the central grey column, internally to the solitary fasciculus. The latter, also, is crossed by medullated bands which run from the grey column outwards towards the substantia gelatinosa and suggest an origin from the bundles afore-described. Both these last and their possible outward prolongations are barely visible in the morbid specimens, although carefully sought for. The application of Pal's method confirms the foregoing.

There is a decided increase of connective-tissue throughout the areas of the funiculi solitarii.

Occupying the periphery of the median portion of the floor of the fourth ventricle, lying behind and in a line with each vago-accessory nucleus, is an area of degeneration, about $\frac{4}{100}$ inch wide and extending $\frac{3}{100}$ inch into the medulla, fairly-defined by its structural features. The vascular elements constituted the most notable details; large channels are seen twisted and variously-contorted. Groups of two to four orifices are visible encased in a thick connective-sheath; the vessel-coats much hypertrophied. Isolated vessel-orifices are the less frequent; their thickened walls are plenteously-besprinkled with connective-tissue cells. Small, round, nucleated granular cells, degenerate nerve-cells, connective-nuclei and leucocytes are freely scattered about this part of the field. This morbid change is found below the planes of opening up of central canal for some little distance, but to a less extent.

The scanty grouped nerve-cells placed postero-laterally to the central canal below the level of the hypoglossal nuclei, which, at lower planes, of inflated pyriform shape (though small and irregular) assume higher up the fusiform character of the vago-accessory cells, are without doubt largely wanting; indicated only by shrunken masses rarely joined by an approximately shapely cell.

The vagal nuclei show degeneration throughout their extent, but not equally distributed on either side. The area of lesion is rather deeply-stained, is very friable, dotted with cell-remnants and wanting the usual fine reticulum afforded by the cell-processes, and everywhere traversed by capillary-trunks; is generally of *débris*-like aspect. Those cells present are in great part shrivelled and apolar, rounded or irregular in contour, seldom fusiform. Are most intact anteriorly. The outer group behind the solitary funicle is similarly affected to the internal.

Quadrigeminal Region.—In sections taken through the lower part of the nates, including a portion of the inner geniculate body and optic root, the latter on the right side shows an excessive permeation by thick-walled vessels, cut in all directions. The following appear free from morbid change—both geniculate bodies, the nuclei of third nerve, at all levels, the nerve-roots, the large cells of the deep layers of the nates, radial fibres of Meynert, posterior longitudinal fasciculi, descending roots and vesicular cells of fifth nerve.

In planes including the fifth descending roots and the fibres which, bordering the central grey matter, run anteriorly into the tegmentum, is an area on the left side extending into the grey substance and outwards to the loop layer of tegmentum, occupied by myriads of small capillary segments. In place of diffuse distribution, the vessels on the right side form a tolerably compact band, lying midway between the fifth descending roots and the proc. e cerebello ad cerebrum, passing with circular sweep outwards towards the entrance of the optic root. No textural change is observed in the neurine-tissue in which placed, and this with the absence of disease in the vessel-tunics and the presence of a dark cylindrical mass in a large trunk, suggests congestion from obstruction by plugging.

Optic Nerves.—Both are lessened in size and somewhat pyriform in section. There is a large connective and vascular increase throughout. The internal sheath of the nerve is much thickened and shows free arterial supply; from it proceed trabeculæ of correspondingly-increased size to the interior, where frequently they equal in size those near the periphery. In every portion of the nerve the neurine tissue is greatly encroached upon, and in the apical extremity the tracts of connective-tissue are in such magnitude and number that the same constitutes but a meagre part of the field. The nerve-structure, too, is here atrophied, blurred, granular and indistinct. The neuroglia cells are increased in size, but not obviously in number. There is usually seen a definable channel lying outside the fibrous septa, its external wall formed by the delicate ensheathment of the nerve-bundles. In this canal appear occasionally, ramifying corpuscles, and from it are traceable other delicate channellings amongst the nerve-tubules, in which similar cells are placed, forming in proximity and communication an early and imperfect septum. Degenerative changes are most intense at the peripheral ends of the nerves. There is marked hypertrophy of the tunics of the central vessels, and the thick belt of wavy connective surrounding them is plentifully permeated by small arteries with much-thickened coats.

Third Nerves.—Appear normal.

Anterior Crural Nerves.—The nerve-tissue is for most part replaced by fatty and connective-tissue, into which, in many places, extensive hæmorrhages have occurred and which, again, is traversed by hypertrophied and engorged vessels. Only a few nerve-bundles are seen in the total section-area of the nerve, but these, excepting some of the smallest, and, also, in no few a decided overgrowth of perineurium, appear healthy. Not seldom small groups of degenerate tubules are scattered in the inter-neurine tissue, sometimes large fat-globules lying amongst them. Fat also invades the sheath of the larger and more healthy bundles of nerves, but is never witnessed amongst the fibrillæ; elsewhere, when in accumulation, shreds of connective-tissue representing the sheath of vanished nerve-funiculi are found amongst. The septa in nerve-bundles of all sizes alike, between the individual fibres, are exceedingly prominent, and in many the proportion of large nerve-tubuli is much reduced.

Sciatic Nerves.—Normal.

Commentary.—The following pages present a condensed account of the various lesions found in the nervous system of the patient whose history is given.

The degeneration of the posterior spinal columns extends from near the extreme end of the cord to the nuclei of the slender and cuneate columns of the medulla. The commissural zones are almost, as wholes, as deeply involved as the rest and at parts quite. There is destruction by sclerosis of the proper nerve-tissue of those bundles lying posterior to the cord in the cauda equina. The vascularity of the posterior columns throughout the lumbo-dorsal regions is immensely increased and the perivascular spaces engorged with granular cells. Sclerosis exists, slightly, in the lateral columns throughout all the dorsal region, confined to the crossed pyramidal tract; the anterior grey cornua and nerve-roots are without apparent change. Degeneration also affects the vesicular columns of Clarke, their supposed representative cell-groups in the medulla, and the vago-accessory nuclei; the nuclei of the gracile and cuneate columns; funiculus solitarius, some of the arcuate bands, and the vagi, anterior crural and optic nerves. In the cortex cerebri an overgrowth of neuroglia in the first stratum and undue pigmentation and disintegration of the ganglionic clusters of motor area.

A *résume* of the symptoms of tabes dorsalis existent in this patient is—ataxia, loss of patellar reflex, lightning pains, gastric crises, girdle pain, some anæsthesia and delayed sensation, Argyll-Robertson pupils, and optic-nerve atrophy.

It is not known for how long a period the ataxy had existed prior to admission. But as death itself occurred about a year after that, and for two months before the fatal event, all locomotion had been rendered nearly, if not quite impossible, it will be apparent that the rapidity with which the symptoms advanced was great.

In many features the morbid appearances are similar to those described by the writer in a former paper on Locomotor Ataxia (BRAIN, April, 1888). In the present case the tendency has been to dwell especially upon the changes in the lower end of the cord. At the extremity of the conus medullarius, where the canal has become T shaped, no distinction between the diseased cord and a healthy one is apparent. The degeneration of those nerve-bundles lying posteriorly to the cord is very marked, and the lesion in the posterior columns grows in dimensions with the increasing area of section and assumption of fresh medullated tracts from the entering nerve-roots. It is possible that these bundles have relation to the anterior crural nerves in which such extreme degeneration was described.

In the earliest sections shewing the spinal canal in its central position, only a narrow tract of medullary fibres exists between the posterior cornua and the investing cord-membranes and betwixt the cornua themselves. The last tract, indeed, is a very narrow one, owing to the replacement by connective-tissue of the inner radicular fibres, so that the grey cornua appear, when Weigert's stain is used, to nearly touch. At higher planes, the portions of the root zones adjoining the entry of the nerve-bundles (which divided at their junction with the cord are seen intensely sclerosed) are sparsely possessed of medullated tubes, and these areas constantly increasing, the latter tubes become more aggregated towards the mid-line. Whether these are represented in the oval-shaped area occupying the posterior half or two-thirds of the mesial septum in the lumbar segment, whose fibres remain intact, the absence of a completely-successive series of sections renders it impossible to decide.

The arterial and periarteritic changes are as marked features as regards the spinal cord in this case as they were in the previously-published one. In both no renal nor hepatic cirrhosis was present; in the earlier case, no symptoms nor history of syphilis were elicited, but in the present one, a fair presumption of the disease having existed may be indulged in.

The degeneration in both cord and nerves was too far advanced to afford evidence as to the histological element in which the lesion started. None of the nerve-bundles in the cauda equina were discovered in an intermediate condition even.

The patient escaped any convulsive seizures; there were no meningeal adhesions found at the autopsy.

It is worthy of remark that although degeneration was present in both vesicular columns and was as widespread in the tracts of Goll and Burdach as could very well be, yet the entire periphero-lateral zones of the cord, which include both direct cerebellar and (so-called) antero-lateral tracts, were perfectly free from lesion, as also the corresponding region of the medulla. Again, the group of medullated tubules placed anteriorly to Clarke's column, and those which, obliquely divided, pass outwards across the pyramidal tract of lateral column, are seemingly intact. Allusion may here be made to the bilateral disease in Clarke's columns, the vago-accessory nuclei and those scattered cell-groups in the lower portions of the medulla, which, perhaps, represent an interrupted connection betwixt the two. This in relation to the gastric crises present.

With reference to the ocular symptoms, no morbid changes were observed except those described in the optic nerves and discs; nor was any difference in degree of lesion found between right and left nerve. The consensual reflex was never quite lost.

The whole cerebro-spinal axis was unusually resistant to aqueous solutions of aniline-dyes, although hardened and cut as soon as possible. Alcoholic solutions stained readily enough. No difficulty was experienced with hæmatoxylin.

ILLUSTRATIONS TO THE PAPER ON THE PERONEAL FORM OR LEG TYPE OF PROGRESSIVE MUSCULAR ATROPHY, BY DR. B. SACHS, NEW YORK.

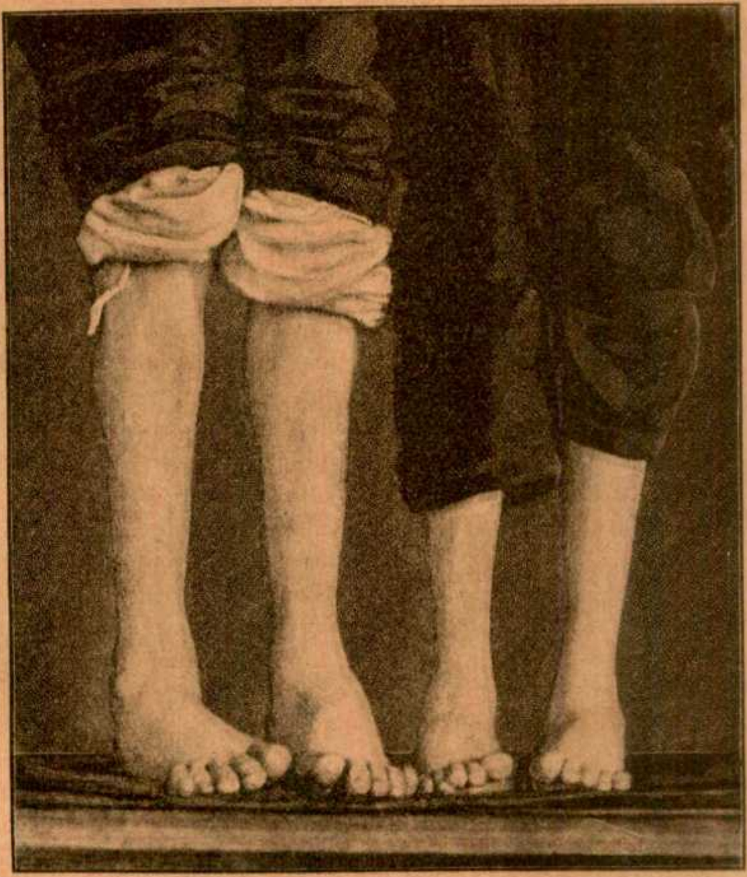


FIG. 1.—Showing condition of feet and legs in both boys, eight months and one year respectively after first operation.

t. 15

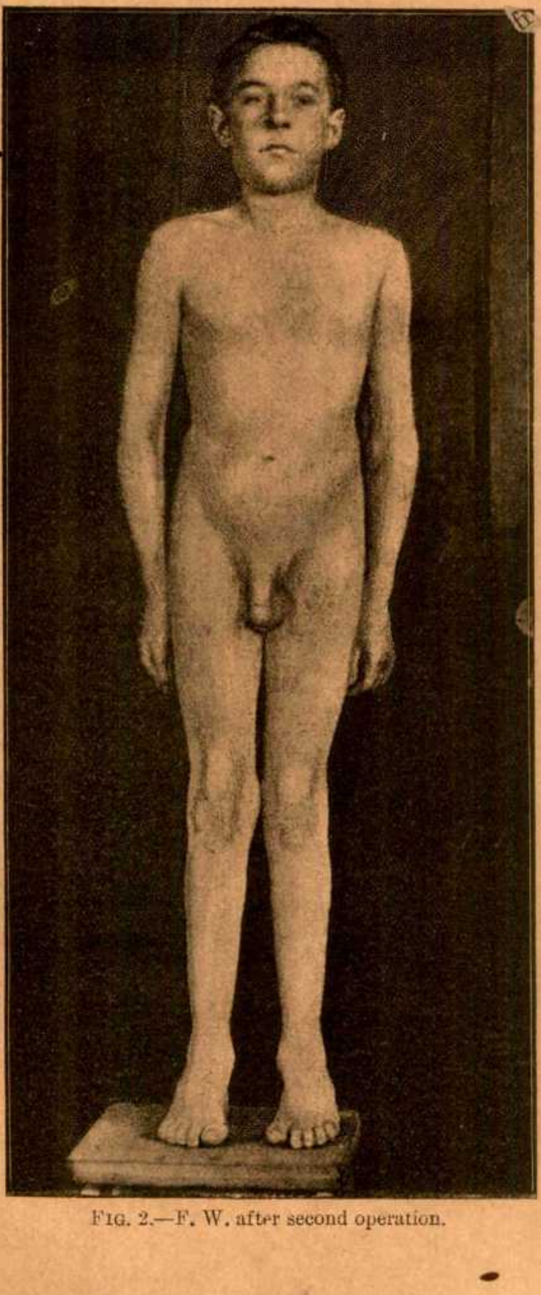


FIG. 2.—F. W. after second operation.

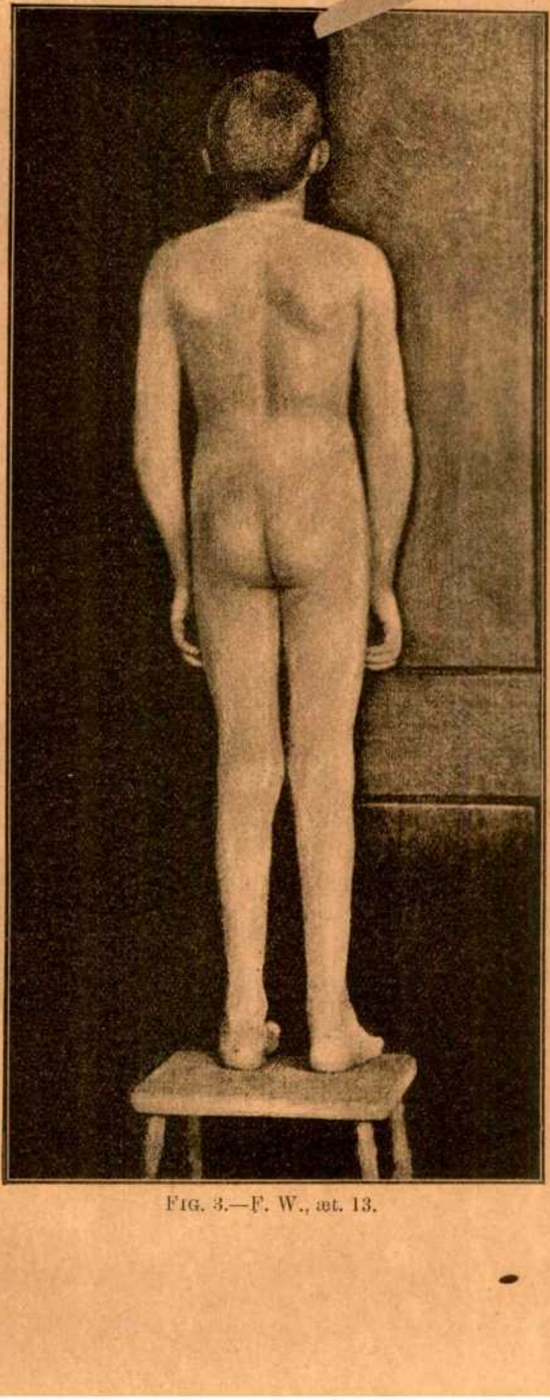


FIG. 3.—F. W., æt. 13.

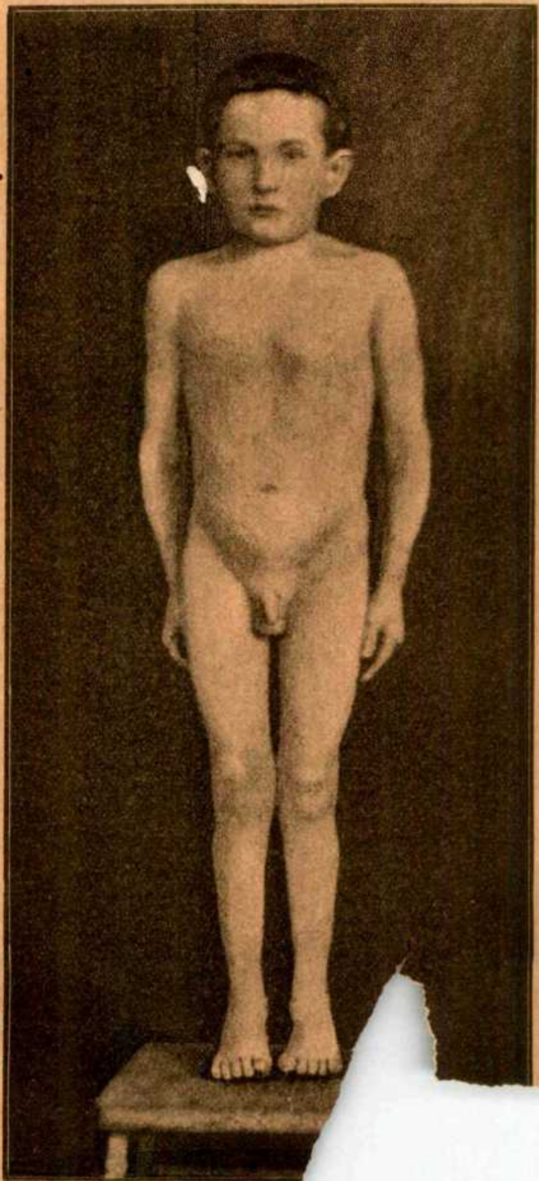


FIG. 4.—After seco

muscles respond to strong currents only; leg muscles, posterior group respond to strong currents in anterior tibial muscle and in extensors of toes and foot; no response on direct or indirect faradic excitation. Galvanic examination limited on account of want of time to muscles below knee. From peroneal nerve first KCC with 13 milliampères (ma.) in extensor hallucis longus; no ACC obtainable with current of 18 ma. Direct excitation of tibialis anticus KCC 14 ma.; ACC 16 ma. Extensor hallucis longus, KCC 13 ma.; ACC 16 ma. All contractions slow. No contraction could be obtained with the strongest currents at command in extensor communis digitorum. In the peronei muscles KCC at 13 ma.; ACC not at 20 ma.

The electrical examination, therefore, shows a diminution in the faradic responses of the *thigh* muscles; the galvanic examination of these muscles was not made at this time, but at a former examination I noted contractions with strong, almost unbearable currents, the anodic being equal to the cathodic effect. In the leg muscles we find that the posterior group alone respond to strong faradic currents, no response in all the anterior muscles, and in these muscles the galvanic examination shows that unusually strong currents are necessary to bring about contractions in the muscles, but that the formula is not altered; that the contractions are decidedly slow. Furthermore we may note this difference from typical poliomyelitis, that the muscles which were in a tolerable state of preservation could be excited by the indirect method fully as well as by the direct. We have, therefore, a partial re-action of [illegible] in most of the muscles of the legs.

Case II.—T[illegible] her was admitted t[illegible] hospital November [illegible] ent [illegible] when [illegible] toes slightly inverted. He returned to the hospital November 12th, 1888, with paralytic limp. The right foot can be everted about 10° beyond the median line and flexed to about 85°. The left can be everted to median line and flexed to 90°. Stands with toes inverted; whole of outer surface rolled outward and downward (see Fig. 1). Double achillotomy was again performed. Division of plantar fasciæ was made. There followed application of Thomas' tarsiclast and plaster splints, with the result as shown in Figures 4 and 5. Two months after the operation it was noted that the patient walks on his feet in typical calcaneus when using his shoes, without apparatus squarely on sole of feet, with disposition of feet to roll inward. He can voluntarily flex ankle-joint a little beyond 90°, but in so doing toes are hyper-extended and the back of foot drops. Still walks with peroneal type of paralysis; marked disposition to pes varus. I had occasion to examine this boy before the second operation and again on May 6th of this year. I give the results of both examinations.

William is of stouter and shorter stature than his brother. Intelligence good. His external appearance peculiar in this that his broad chest and fat stomach are in curious contrast to his spindle-shaped extremities. The circumference of chest 26 inches; right arm, 6½ inches; left arm, 6¼ inches; right forearm, 6½ inches, left forearm, 6¼ inches. Grasp of both hands very weak, cannot move dynamometer more than two degrees. A general emaciation of all parts of upper extremities. Very distinct atrophy of infra-spinatus. In the legs, general atrophy is very well marked: right thigh (4 inches [illegible] lla), 11 inches; left thigh, 1[illegible] inches; right calf, [illegible] calf, 8⅜ inches. [illegible] dling [illegible] difficulty in [illegible] raise [illegible] aise

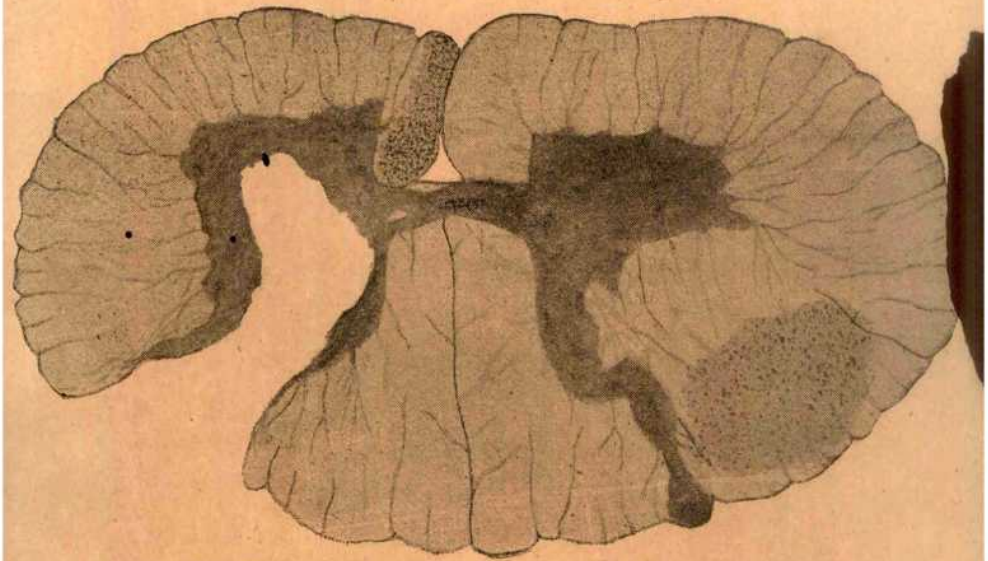
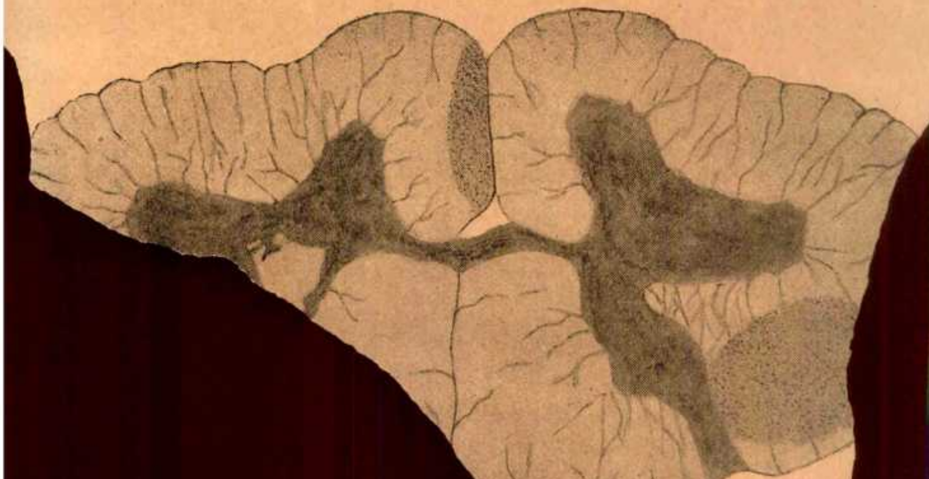


FIG. 3.—Cross sections through the spinal cord. Low power.



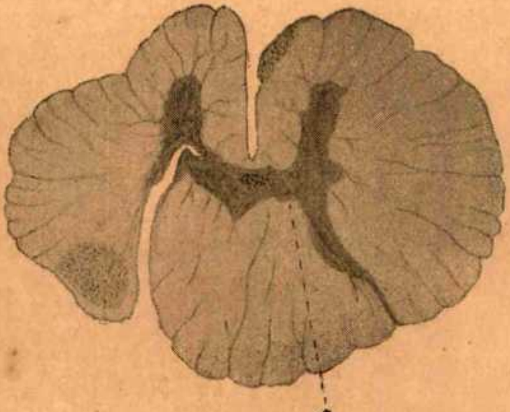


FIG. 7.—A. Hyaline mass in commissure, around which hæmorrhage has taken place. Cross sections through the spinal cord. Low power.

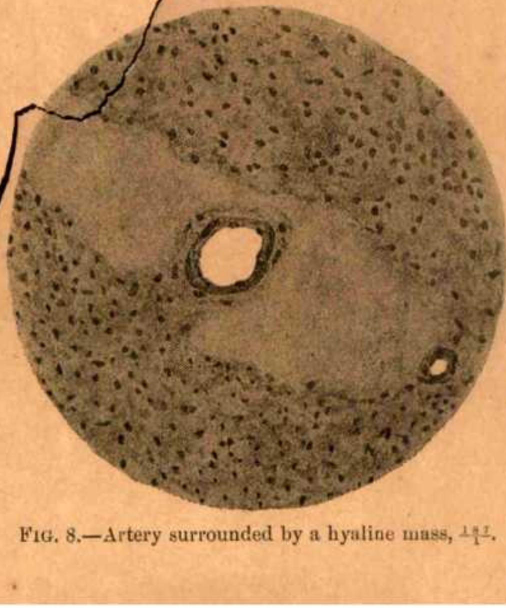


FIG. 8.—Artery surrounded by a hyaline mass, $\frac{187}{1}$.

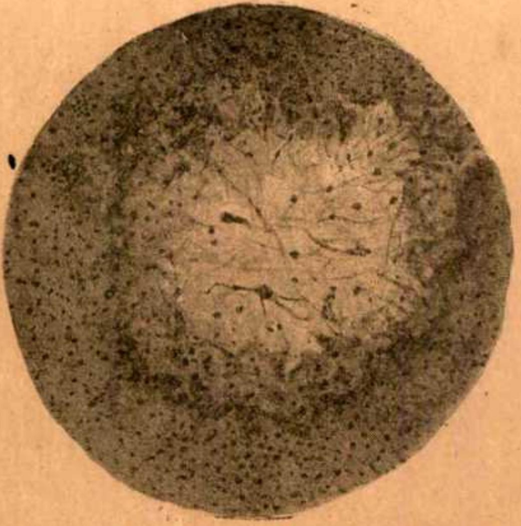


FIG. 9.—Hyaline mass from the intermediate tract, showing the coarse fibres crossing it, with the scattered nuclei, $\frac{180}{1}$.

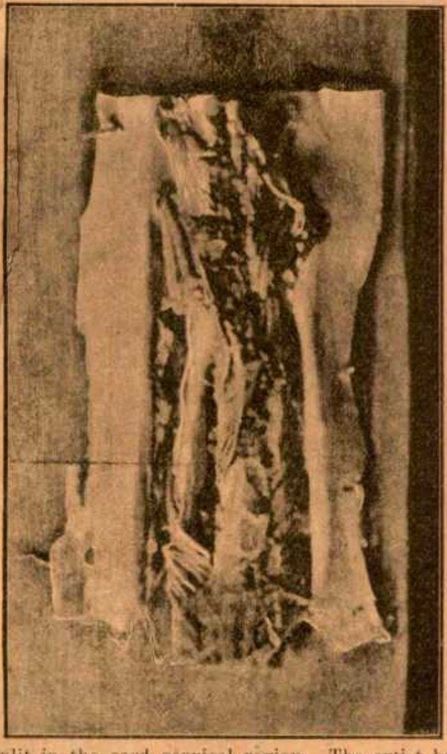


FIG. A.—The split in the cord cervical region. The artist has reversed the sides of the cord in Figs. 1, 2, 3, 4 and 5, the sclerosed indirect pyramidan tract being on the left side.

Autopsy 14 hours after death.—Cadaver of medium build. Rigor mortis not well marked. Nutrition except of paralysed muscles good. No joint disease. Contraction of arm and leg still persistent. Arm and leg of left side distinctly smaller than their opposites.

On removing the skull cap, as soon as the saw had cut the veins of the diploë, dark blood in unusual quantity flowed from the openings. The skull was of ordinary thickness, without noticeable asymetry. Upon the parietal surface of the dura mater, near the postero-superior angle of the right parietal bone, was a recent agglomerated ca[illegible]morrhage, 4½cm. in length by 1½cm. broad, surrou[illegible]arged Pacchionian body. The long diameter was [illegible] the inter-hemispheric fissure. The dura upon the [illegible] vessels highly injected. The sinuses [illegible]agulated blood.

was now plugged and the body turned on its face, the spinal canal opened and the medulla spinalis examined *in situ* by slitting up the dura mater. Recent clots and fluid blood covered the entire surface of the pia in the arachnoid space—the coagula being more common about the cervical enlargement than in the other regions of the cord. Opposite the third, fourth, and part of the fifth cervical vertebræ, the pia was seen to be ruptured along the right posterior nerve roots. Further examination showed a split in the substance of the cord, running in the direction of its long diameter, a little over 3cm. in length, and extending apparently as far forwards as th[illegible]r grey horn. The break was lateral to the right poste[illegible]ots, just at the point where the sulcus lateralis dors[illegible]its deepest part. A small cavity [illegible]e fissure proper, and

The arterioles are surrounded by vast perivascular spaces filled with hematoidin crystals and *débris* with leucocytes in large numbers. Around these spaces the neuroglia is quite granular, extending in a ring around the opening, while around other spaces the density is only in spots on the space margin. In each case the change extends only a short distance into the surrounding tissue. These alterations are best seen in the subcortical white substance. The changes in the arteriolar walls, which are very slight, are confined principally to the adventitia (slight cell proliferation). No distinct alterations can be made out in the finest capillaries.

In the cortical gray [illegible] the ganglion cells vary in different parts even of the same [illegible]ere and there are portions of cortex that show an [illegible]ly normal state of the cell elements in all the l[illegible] in other parts of the same section not a millimet[illegible]re is almost total destruction cells are to be found, and these are filled with pigment. Totally destroyed cells are excessively rare, the body being simply filled with yellow granules. The axis cylinders in the radiating and other fibres passing through the capsules are normal with straight contours. In some of the bundles crossing the ganglia adjacent to the capsules, the number of the nerve tubes seems unusually small compared with the size of the bundle. The neuralgia is decidedly more granular than in normal specimens.

Medulla Oblongata.—Sections of the medulla were made from the region immediately subjacent to the pons down to the decussation of the pyramids. The nuclei of origin on the floor of the fourth ventricle, the outer, middle [illegible]ner olivary bodies, and the scattered ganglionic masse[illegible] the raphe were normal in every respect. The roots o[illegible] and twelfth nerves as they pass from the medullar[illegible] the ascending root of the trigeminal, the corpora re[illegible] the fibres crossing the greater. The fungoid granulations covering the nuclei give, when fully developed, a very curious appearance to the cells.

In the upper lumbar tract, the region of the second split, the cord exhibits considerable irregularities of formation, the gray parts being especially involved. The left posterior horn is much less developed than the right. Heavy irregularly-shaped masses of gray tissue containing groups of multipolar cells scattered everywhere through it, form the commissure (Fig. 7). The second split is on the side opposite to the cervical one, and commences ventrally near the middle of the anterior horn with a mesial branch, containing many torn bundles of tissue extending into it and covered with clumps of blood cells without *débris* (Fig. 7).

The split in the posterior horn follows very closely the middle of the column, and is separated from the pyramidal tract by a broad band of gray matter, the blood having made its exit near the mid-part of the horn. At the most ventral part of the fissure in the anterior horn, is an irregularly circular area, which takes up little carmine, and is seen on examination with higher powers to be densely packed with red, and less numerous white, blood corpuscles. About th[illegible]ction of the dorsal and mid-third of the horn is a spot of t[illegible] form[illegible]ning several vessels with thickened w[illegible]re is no sign of any hyaline degene[illegible]ration as a whole, indeed, is ra[illegible]l, the only other

Hyaline deposits are frequent in the commissure and mid-parts of the horns, and always have a vessel in their immediate vicinity. Venous thrombi are occasionally met with. No deposits are seen in the white matter. The meningitis is more marked along the posterior than around the anterior columns, though nowhere extensive. The nerve roots are to all appearances perfect.

The vascular system offers considerable variations in the different columns of the cord; in the sclerosed areas there is great proliferation of nuclei in the adventitia; veins of large calibre predominate in numbers over the arterioles; the media has an extremely glassy appearance, in which no fibres of a muscular nature can be seen. The intima shows a slight increase of nuclei. In the other white tracts the vessels have a nearly normal aspect. In the gray columns the nuclei of the intima are increased in numbers, the muscularis is somewhat thickened, the adventitia is normal, or in those vessels surrounded by the hyaline product it is sometimes almost lost, the hyaline matter penetrating between adventitia and muscularis to such an extent as partially to destroy the former, or to push it away from the media.

The reticular s [illegible] ents [illegible] e same character throughout the cor [illegible] ng a little denser in the lumbar reg [illegible]

The comp [illegible]

marked, but in the pons no focus of disease could be seen on transverse section—at least as far as the only examination permitted by the unsatisfactory state of the organ, revealed to the eye. Microscopic examination of the region of the internal capsules gave only the very indefinite appearance of the bundles passing through the ganglia adjacent to the capsules, containing a small proportion of fibres in comparison to the size of the bundle. The cortex furnishes the only definite change found in the nervous elements of the brain—a marked granular destruction of the cortical cells. Unfortunately, again, only a part of the motor region was preserved, and we do not know if this degeneration was general.

Nevertheless, in th [illegible] r region [illegible] sponding to the part which innerva [illegible] alytic [illegible] egeneration is well marked, so m [illegible] t m [illegible] sts are almost totally destroyed.

have no degeneration of the bulbar nuclei, and the paralysis does not begin in the cervical region. The degeneration of the ganglion cells in the anterior horns, though considerable in both the cervical and lumbar enlargements, is pretty equally diffused over both sides. On the other hand, however, we have the cortical degeneration, frequently met with in amyotrophic sclerosis, and which Marie [37] declares to exist in all cases of this affection.

But sclerosis of one pyramidal system is not known even to exist, unless we assume its occurrence in a few cases of Brown-Séquard's paralysis,* and I offer this hypothesis only in the absence of any other possible explanation of the etiology of the hemiplegia.

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complained o and ing in the paretic
leg. Now in t larg . 7), and extending,
too, for some the diameter, we found

[10] Lyster, *Lancet*, ii., 694, 1880.
[11] Page, *Lancet*, 1880.
[12] MacMunn, *Dublin Journal Med. Sciences*, lxix., 1880.
[13] Jendritza, *Allg. Zeitschrift für Pyschiatrie*, xli., 1884.
[14] Chaffey, *Brit. Med. Jour.*, ii., 1885.
[15] Sinclair, *Lancet*, ii., 1885.
[16] Bouchard and Loison, *Jour. de Scien. Med. de Lille*, viii., 1886.
[17] Leyden, *Zeitschrift für Klin. Med.*, xiii., 1887.
[18] Saccheo, *Rev. Med.*, 1836, t. iii., cited by Hayem.
[19] Clanbry, *Jour. Gener. de Med.*, 1808, cited by Hayem.
[20] Colin, cited by Hayem.
[21] Weber, *Jour. hebd.*, t. iv., 1829.
[22] Page, *l.c.*
[23] Jendritza, *l.c.*
[24] Von Recklinghau[illegible] *er Allg. Path.*, 1883, s. 405.
[25] Anna Baumler, *D*[illegible] *Klin. Med.*, 1887.
[26] Hutin, *Nouv. Bil*[illegible] p. 159, cited by Bäumler.

LARYNGISMUS.

BY WILLIAM GAY, M.D., M.R.C.P.

COMING within the provi[illegible]he laryngologist, the general and children's physic[illegible] neurologist, laryngismus has been regarded fro[illegible]oints of view, and has received such an amount [illegible]hat clinically little times in his thirty-four cases of laryngismus. It is possible that the statistics obtained from Great Ormond Street Children's Hospital (West, Gee's and my own) overestimate the frequency of rickets, for it is the exception there to find a child who does not present some of the features of that disease. Some of the discrepancy may probably be accounted for by the inclusion in one list of slight cases of rickets which would be ignored in another. It is to be remembered, however, that laryngismus does not necessarily fall most heavily upon those who have the most marked signs of rickets. Indeed, in some of the most severe cases I have seen, the only evidence of rickets consisted in beading of the ribs, delayed dentition, and a too patent fontanelle. Similarly laryngismus is very often conspicuous by its absence in those cases of rickets in which great thoracic deformity is the most striking feature. This is an important fact in connection with the theory entertained by Hughlings Jackson, and will be reverted to later on. After the publication of his paper I took notes of all cases of severely collapsed chests coming under my notice at Great Ormond Street during the laryngismic season, and not one was complicated with laryngeal spasm.

This season was independently brought into prominence by Gee and Flesch,[1] though it had been noticed casually by some preceding writers and even by Hippocrates,[2] who, writing of the asthma of children (= probably laryngismus), says, "It occurs during winter in cities, whose confined situation precludes them from due exposure to the evaporating influence of the sun and winds," &c., &c. Of sixty-three cases observed by Gee during a course of three years, fifty-eight occurred from January to June (inclusive), and five only from July to December. This curious and important fact he interprets by supposing that "the weather necessitates the keeping of children indoors, and may be in one room for a long time, thus begetting a nervous erethism, which shows itself as a spasmodic diathesis." In very many cases such a history can be obtained, and the following case is a sufficiently striking illustration of it. A girl of six (the only living child) was brought to Great Ormond Street with severe laryngismus, which recurred each winter. On inquiry I found that she was kept in one room, swathed in flannels, with a fire night and day, and a screen in front of the door. In this way she was, as it were, hermetically sealed up for the whole of each succeeding winter. A "due exposure to the evaporating influence of the sun and winds," coldish baths and cod-liver oil soon relieved her of her laryngismus, and as the hygienic precautions were continued throughout the next winter, she suffered no relapse. An older brother, who had been coddled in the same way, died in an attack of laryngismus—a victim probably to misdirected solicitude. Very generally the same tale was told, "we thought it better to keep the children indoors in case they caught cold." So they were kept indoors and very often remained there the whole winter.

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[1] St. Bartholomew Hospital Reports, vol. iii., 1867.

[2] Hippoc., *Fœsii Francof.*, 1621, p. 281.

To what extent laryngismus is directly hereditary is of course difficult to determine, but it is common to find that its subjects are derived from a very neurotic strain. In eighteen of my own cases (fifty) there was ample evidence of such a neuropathic heredity. It sometimes happens that at the appropriate age, each member of a family is attacked with laryngismus. I have myself met with instances of this, but none so striking as those recorded by Reid[1] and Gerhardt.[2] According to the former authority, out of a family of fifteen only one escaped, and in ten the disease proved fatal, and the latter records an instance in which seven children were attacked out of a family of nine. In such cases there is probably a strongly marked neurotic heredity, and the children are all brought up under the same faulty hygienic conditions.

The most usual age at which the disease first occurs is from the seventh to the twenty-fourth month. Cases commencing some months before this should be regarded with a certain amount of suspicion, especially those dating from birth, of which an instance is recorded by Mackenzie[3]. On

[1] *Lancet*, May 1st, 1847. [2] *Lehrbuch der kinderkrankheiten*, 1871, p. 285.
[3] "Diseases of Larynx," vol. i., p. 480.

the other hand it is not at all uncommon for laryngismus to recur every winter for four or five years, and sometimes even longer. As a rule more boys than girls are attacked; Steffen, from a large number of statistics, puts the proportion at two to one. My own figures, twenty-six females and twenty-four males, are too few to invalidate the general conclusion. No reason has yet been suggested to explain this greater proneness of males to the affection, but Hilton Fagge[1] recalls the fact that there is the same peculiar liability of males to tubercular meningitis, and regards the two observations as equally inexplicable.

There are many exciting causes. Paroxysms are common at any time [illegible] but especially so an hour or two after the ch[illegible] put to bed, or in the early morning. A pas[illegible] almost [illegible] to bring on an attack in a laryn[illegible] less surely.

laryngismus. The spasm may be incomplete, so that sufficient room is present between the more or less closely adducted vocal cords to allow of the passage of air. In this case, there is a mere croupy sound with each inspiration, which produces only slight cyanosis and troubles the child but little. Older patients of from five to ten years sometimes experience a choking sensation on swallowing or crying, but there may be no croupy sound and only slight cyanosis.

A clue to all these cases is afforded by the contraction of certain facial muscles on mechanical irritation of the facial nerve branches—facial irritability.[1] Dr. Abercrombie was accustomed to point out to his students, when I first became his clinical assistant at Gr[illegible] Street, that on smartly brushing the forefin[illegible]r so to the outer-side of the exter[illegible]angle of [illegible]ontraction of the orbicularis pa[illegible]su[illegible]laryngismus and

It will be well now to briefly notice some of the many theories concerning the nature of laryngismus. Several of the older writers, *e.g.*, Rush[1] and Clarke,[2] maintained that the asthma of children consisted essentially of a convulsion, and many modern writers would be inclined to agree with this view of the case—as far as it went. Kopp,[3] in 1830, restarted the enlarged thymus theory, which had been previously suggested by Richa and Vendries a little more than a century before; but this received its *coup de grâce* from Trousseau,[4] who showed that no enlarged thymus was commonly found in cases of death from laryngismus, and that if enlarged, it was not necessarily attended with laryngismus.

In 1836, Ley[5] put forward the idea that enlarged glands in the neck and mediastinum were the source of the affection. This view is not yet extinct, but taught in a modified form by Goodhart and Steffen.[6] The former considers that the laryngeal spasm is of central origin when rickets is well marked, but that those cases, in which rickets is not so striking a feature and there is cough with a laryngeal tone, bronchitis, or hoarseness, or all combined, are due to reflex irritation, arising from worry of the vagi terminations by enlarged mediastinal glands. There does not seem to me sufficient evidence to justify such a sharp distinction in the pathology of different cases of laryngismus. They vary very much in degree, it is true, but a slight paroxysm induced by passion is as clearly of central origin as that is reflex, which results from exposure to wind or colder air in more severe cases. In fact, in every case some of the paroxysms are no doubt centrally ordained, and others are as certainly of a reflex character. Steffen does not regard the enlarged gland theory as universally applicable, and evidently adopts it, *faute de mieux*. He suggests that the paroxysmal nature of the affection depends upon circulatory changes, which increase or diminish the size of the glands, and so also their pressure on the nerves. This explanation is constructed out of very problematical materials, requires a wonderfully fine adjustment of gland to nerve, and fails altogether to account for certain phases of laryngismus. Why, for instance, should the glands swell to the required extent on exposure of the body to a cold atmosphere, on emotion or swallowing?

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[1] 'Dissertation on the Spasmodic Asthma of Children,' 1770.

[2] 'Commentaries on the Diseases of Children,' 1815, p. 87.

[3] *Denkwürdigkeiten*, 1830, p. 1.

[4] *Clinical Med.*, *Sydenham Soc. Trans.*, vol. i., p. 353.

[5] 'An Essay on Laryngismus,' London, 1836.

[6] *Loc. cit.*

Marshall Hall[1] was not long in applying his reflex theory to the elucidation of laryngismus, and although he pushed it too far, I am not at all sure that he did not recognise that some cases were of central origin. He arranged the reflex arc in schematic form, thus:—

Excitors.	*Centre.*	*Motors.*
Trifacial (dental)	Medulla	Recurrent of vagus
Vagus (gastric)		Intescostals
Spinal (intestinal)		Diaphragmatic.

This was a distinct advance upon previous efforts, and may still be accepted as a sufficient explanation of many of the paroxysms.

Elsässer[2] believed that laryngismus was accompanied with craniotabes, which allowed of pressure on the brain. This theory stands discredited for very much the same reason as Kopp's.

Probably no one would be found now-a-days to subscribe to Hood's[3] opinion that laryngismus was due to an enlarged liver impeding the descent of the diaphragm.

Hughlings Jackson has been the author of two theories in regard to laryngismus. In the first,[4] he regards it as a partial convulsion—partial, "because of the imperfect union of different sections of the nervous system." He has since discarded this in favour of a much more elaborate theory,[5] which is difficult to reproduce in a few words, but amounts practically to this: that the natural stimulant of the respiratory centre is venous blood, and that if for any reason there

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[1] 'Diseases and Derangements of the Nervous System,' p. 71.

[2] *Der weiche Hinterkopf*, 1843, p. 161.

[3] 'On Scarlet Fever and Crowing Inspiration,' 1857.

[4] Reynold's 'System of Medicine,' vol. ii., p. 229.

[5] *Brain*, April, 1886.

"lowest level," but must profoundly influence and tend to retard the developing higher functions, including those of inhibition. Hence would ensue an exaggeration of that "excitability," which is the characteristic of even a healthy infant's brain. If this be so, we should expect that in rickets, which is essentially a disease of mal-nutrition, the centres (being inadequately controlled) would be in a state of very unstable equilibrium, and that the frail barrier of restraint would break down under conditions which would be easily resisted by a healthy brain. The frequency of convulsive affections in rickety subjects shows that this indeed is the case. In many cases of severe rickets the development of the brain is evidently retarded—at times so much so that it may be extremely difficult to determine that the child is not really an idiot. In some of the slighter cases of rickets, however, a certain precocity in the evolution of some of the centres is noticeable, but their instability is generally equally apparent. This, I take it, depends on the development of the centres ahead of their checking arrangements to a greater degree than obtains in a healthy brain. In other words, I believe that the chief effect of rickets upon the infant's brain consists not so much in retarding the development of the centres, as in diminishing their inhibition and therefore their powers of resistance.

It has been previously noticed that laryngismus is generally accompanied with an increased excitability of the motor nerves. What the exact significance of this is one cannot, at present, determine. It is inconceivable that such an inert tissue as the fibrous coverings of the nerves should develop an irritability communicable to the axis cylinders; besides, the substance of Schwann bars the way, and if this were rendered excitable, the transmission of impulses along the nerve would become incomplete or impossible on account of their diffusion. The increased irritability therefore must lie in the axis cylinders themselves. Since there is no morphological distinction between afferent and efferent nerve fibres, it is quite possible that the axis cylinders of the sensory nerves participate in the increased excitability of the motor. If this be so, it is evident that the reflex activity of the individual is increased not only in extent but in degree, for the normal intensity of the afferent impulse will become exaggerated by the increased irritability of the sensory nerves, and hence a correspondingly more complete discharge of the centre will take place, for as Mercier[1] says, "a stronger shock will displace more atoms than a weaker shock; and since the greater number of atoms displaced, the more force is liberated, it follows that, other things being equal, the greater the disturbing force, the more powerful will be the resulting discharge." This effect will tend to be further increased by the greater instability of the out-going motor fibres, on account of which a lesser stimulus than normal is required to produce the same result. The same is no doubt also true of central impulses, the intensity of which, to produce a given result, would require to be in inverse proportion to the excitability of the motor outlets; or, otherwise expressed, it is necessary for the inhibitory arrangements of the centres to be the more complete, as the facility for the trajection of impulses to the periphery is the more pronounced, *i.e.*, in proportion to the degree of motor excitability. But reasons have been brought forward to show that the inhibition of developing centres is incomplete, and it has been suggested that the occurrence of rickets tends further to accentuate the deficiency. Many children have moreover a strongly marked neurotic predisposition, and this, with Clifford Allbutt,[2] I regard as merely a tendency to a congenital defect of inhibition. For these several reasons the brains of rickety children, especially those of a neurotic strain, are unstable—deficent in the power of resistance. This, however, may be quite sufficient for the proper regulation of the cerebral processes, provided only no great demand be made upon it, and the child be placed under favourable conditions of existence. If these necessities be not complied with, it is not surprising that a breakdown takes place, which may be only partial and limited, or complete, and resulting in general convulsions. It is clear that the evidence of this widespread deficiated act—would proceed to the œsophageal centre—one of the most stable in the whole nervous economy—and to the laryngeal—which has been shown to be just the reverse. The œsophageal element is therefore efficiently performed, but the laryngeal is "overdone" (if I may so express it); for the control of the centre, deficient from the first, has probably been still further weakened by frequent discharges, the result of emotion, and the simple contraction of the adductor muscles of the cord is replaced by a spasm. This, as far as my own experience goes, is always slight, as would be expected, and never attains the profound degree which may mark the laryngeal spasm arising from emotion. I have seen one case in which I suspected that the œsophageal centre was equally unstable with the laryngeal. The patient was under the care of Dr. Bristowe, who kindly allows me to mention the case. He was a boy of about four years old, who had occasional attacks of œsophagismus—one of which was so severe that he had to be fed for some time with nutrient enemata. No obvious obstruction could be detected by means of bougies, and at times he was perfectly well. He suffered severely from croup each winter, and had easily obtainable facial irritability when I saw him. It seemed to me that, for some reason or another, the centre for swallowing was equally, with the laryngeal, under deficient control, and that the action of the œsophageal muscles was "overdone" for a similar reason to that which has been previously suggested in regard to laryngeal spasm.

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[1] 'Nervous System and Mind,' p. 25.

[2] *Med. Times*, February 14th, 1885.

If the views here enunciated be correct, it is evident that the paroxysms primarily result, not from any increased excitability of the lowest level centres, but from a deficiency of their controlling arrangements, situated at a higher level. Dr. Jackson believes that the seat of the discharging centres is in the lowest level. This may possibly be the case, although it would seem more likely that these, being the first developed, would be more fully controlled, and therefore less unstable, than their developing cortical representatives, and Dr. Semon, writing to me on this subject, says that for several years he has thought and taught at St. Thomas's Hospital that laryngismus stridulus is a cortical affection. It is possible that future experiments on the cortical localisation of the respiratory muscles will be of great assistance in helping to clear up this question.

Diagnosis.—The only real difficulty is experienced in cases of recurved epiglottis, which may closely simulate laryngismus. They are, however, congenital, and a certain amount of constant inspiratory stridor may be heard, if carefully listened for. They are not necessarily associated with rickets, and the presence or absence of facial irritability will be here of some service.

Prognosis.—Larnygismus is certainly not now the fatal disease it was once considered. The statistics of the older, and indeed some of the comparatively modern writers, would place the mortality at 50 per cent., but with such a terrible affection we have happily not now to deal, at any rate in this country. The prognosis is generally good, especially when it is possible to place the patient under proper hygienic conditions and the emotional tendency is not pronounced. A child who is subject to uncontrollable fits of passion may at any time die in a laryngismic paroxysm, as indeed happened in the case before narrated.

Treatment.—From what has gone before it is evident that the chief factor in the treatment of laryngismus consists in placing the patient under favourable conditions of existence. There must be no " coddling." The child must be fed on rational principles, and allowed to breathe pure air. If this cannot be obtained in a town, the patient should, when possible, be taken to live in the country, and permitted to respire as much fresh air as he can get. This alone, as I have frequently found, is attended with most excellent results. One case especially recurs to my mind of a female child, who was treated to no purpose for months with all the most approved pharmacopœial remedies, but whose opportunities of obtaining pure air were *nil.* The paroxyms were exceedingly severe, with carpo-pedal contractions and occasional convulsions. I advised her removal to the country, where she rapidly got quite well, although the laryngismic season was then at its height. When the affection is so severe that the slightest exposure to cold air brings on a paroxysm, it becomes necessary to gradually educate the patient to be able to resist all kinds of weather. A commencement should be made with an occasional move from one room to another, then a venture may be made out of doors, when the day is sunny and free from wind, then colder days may be chosen and so on. A valuable accessory to this line of treatment consists of a morning bath, with a sort of spinal douche, at first of tepid water, subsequently rendered more cold. By means of both of these measures the child's capability of resisting external influences is vastly increased. Of the large number of vaunted specifics, there is really only one worthy the name—cod-liver oil. This is not only the best treatment for the rickets, so frequently present, but is, I believe, of the greatest possible value in assisting to "build up" the brain. I therefore prescribe it equally in all cases of laryngismus, without any reference to the intensity of the rickets. Chloral, bromide of potash or of ammonia may be given at night time, especially when there is spasmodic contraction of the muscles during sleep, or the nocturnal paroxysms of laryngismus are severe. If the case is complicated with tetany or convulsions, of course additional means of treatment will have to be adopted, but the question of the hygienic environment of the individual is still of the first importance.

The materials for the construction of this paper were obtained during a three years' clinical assistancy at Great Ormond Street Infirmary for Sick Children with Dr. Abercrombie, to whom I would express my deepest thanks for allowing me to use them as I have done, and also for much kind assistance generally.

Distinctions were laid down as marking the differential diagnosis clearly, and so they did if one compared extreme typical examples of melancholia attonita and acute dementia (stuporose insanity). Nevertheless many cases of the great group presented strange anomalies, failing to agree with either of the types named, and further division was obviously needed. Some years ago it was a project of the writer's to analyse a large number of cases in pursuance of an endeavour to arrive at that division. But special reasons intervened. Amongst other chief difficulties confronting one was the placing, nosologically, of the more marked of those cases which have been termed examples of *katatonie;* for although it had been first described several years previously, yet at that time a knowledge of it had not yet filtered through to general cognisance in this country; a brief paragraph or two which appeared in medical journals here had not arrested attention strongly, if at all, and in fact were not very enlightening.

Further sub-divisions have been made, other sub-groups being carved out of the mass, but it is doubtful whether the cleavage has been on lines of natural fission. From the first, the names designating individual subdivisions of the large group have been variously used by different writers, the same names or their synonyms being applied to sub-groups differing more or less as to their constituent elements. Therefore we find a variety of terms, often not similarly applied, and making separate mention necessary:—idiotisme (Ph. Pinel), stupidité, démence aiguë, acute stupor of the insane, mélancolie avec stupeur, melancholia attonita, melancholia with stupor, acute dementia; acute primary or primary dementia; œdema cerebri, stupor, cataloptoid insanity, catalepsy, ecstasy, stuporous insanity, mental stupor, acute primary dementia with stupor, of the varieties attonic, post-maniacal, stupid-hallucinatory, and stuporous form of acute para[illegible]allucinatory- or psychic- or pseudo-stupor. Also [illegible]e, hebephrenia, some of the cases classed as mas[illegible] insanity; incipient general paralysis with stupor[illegible]oms. Apart stand cases such paralysis, and so on, which are not to be discussed here, and which belong to the great pathological states of which they are incidental accompaniments and dependencies

Although Kahlbaum, who named *Katatonie*, and who first described it as a separate malady, demands for it an application far wider than can be admitted, there certainly is a set of cases forming a sub-group in which the clinical phenomena and general course of the disease are so distinct that it will be necessary to take that case-group into account and to concede to it a separate existence as at least a variety or sub-form—if not as a separate and distinct form of mental disease, for which last view there is much to be said. At least as a symptom-assemblage or syndrôme, the name katatonie will have its uses in mental medicine.

As to its nosological position, I placed katatonie as an appendix to the class of psycho-neuroses, as one of which it has been described by others, but pointed out that there were elements in katatonie which led one to consider it equally well placed in the second great group of mental maladies (as I classify), namely that in which hereditary influences predominate, and consisting of psychoses transformed from ordinary neuroses, of psychoses of hereditary or other morbid constitutional neurosal or mental degeneration, of defective organo-mental constitution, or of incomplete or arrested brain-development. Here it might stand between the class of periodical and circular psychoses on the one hand, and on the other the adjoining class containing, *inter alia*, hebephrenia, paranoia and simple hereditary insanity. This is not the view of Kahlbaum. But having expressed this view for two or three years past in briefly describing *katatonie* in lectures delivered, it was gratifying to me to find by their contribution in the last number of BRAIN, that a partly similar view is taken by Drs. T. Séglas and Ph. Chaslin.

Little being known of *ka*[illegible] this country, I would have preferred to write delib[illegible] with full records of cases, but until the eleven[illegible] had no intention or thought of writing; and the [illegible] of time at disposal tion to a reproduction from my lectures above mentioned of a brief clinical account of the condition, to which the notes of an illustrative case are now added.

The following is the reproduction from the lectures:—

Katatonie.—This was first described by Kahlbaum in 1874. It is usually placed in the sub-groups called psychoneuroses, but it might well be placed in the second great group (as I classify), namely the group of hereditary neurosal or mental degenerations, where it might take a place adjoining the sub-group consisting of the periodical and circular mental diseases, between it and the next sub-group made, *viz.*, that in which the chief constituents are paranoia, hebephrenia and the hereditary psychoses marked chiefly by impulse or by moral perversion. For it is essentially somewhat cyclical in nature; shows a great tendency to change from phase to phase, often returning to a previous one as if by a sort of relapse; is not so curable as supposed by Kahlbaum; the subjects of it often are members of families showing hereditary psychic degeneration; and, should death follow, the brain-atrophy, anæmia and marked basal meningeal changes (or the passive hyperæmia and œdema of meninges) indicate a preceding profound alteration of nutrition of brain, and that in comparatively young subjects, for katatonie chiefly affects the relatively young. There is a tendency also to pulmonary phthisis, which is particularly connected with some phases of the disease, namely the stuporose-cataleptic. With some phases of the disease, we say, for the malady manifests itself in periods or phases in a more or less cyclical manner, however irregular and defective this circular aspect of its course may be.

Often the initial stage or first period is one of emotional *depression*, one superficially wearing a part-likeness to ordinary melancholia. Muscular tension or spasmodic movements, grimaces and contortions, or choreoid facial movements, or hysteroid or epileptoid convulsions occur in this period, and are often found in subsequent periods; or instead of them more constant rhythmical movements about the face or limbs.

The delusions, self-accusations, or fears, in the depressed period may concern masturbation, the commission of "the unpardonable sin," the imagined poisoning or suffocation or bodily injuries inflicted upon, or persecution of, the patient. Suicidal attempts or inclinations may occur, particularly in connection with the hallucinations. For hallucinations are, at least often, present, are usually of depressed character, often indeed of a character that should be terrifying to the patient; but it is noteworthy that sometimes he does not appear to be profoundly dejected or terrified; his half-silly grimaces and even occasional hilarity belie the purport of his delusions and hallucinations. Occipital headache may be marked.

In some cases there is an early period of religious ecstasy, with all the features of that condition.

Following this disturbed time of depression usually comes an *excited* period. And now are often expansive ideas and feeling of the religious or of the social order, and much gesticulation and protracted emphasised elocutionary displays; also a disposition to repeat phrases or words, to emphasise some particular words or phrases, to make an oration of words or part-sentences without meaning or order (verbigeration); a set harangue or discourse, or solemn, pompous, important, and yet of fragments of sentences, repeated or disconnected; or to repeat unintelligible words or syllable-successions not belonging to any language; and all this with much gesticulation, muscular tension, effort, emphasis and forcibleness; and often facial grimaces or spasms, or rhythmical movements, or irregular ones, of the extremities; with a tendency to contradict, to oppose, to resist everything; to refuse food; to resist being washed, dressed or raised from bed. The muscular tension may represent the delusion in terms of gesture—attitude—physiognomy—expression or language; the morbid thought being (so to speak) moulded in outward non-vocal expression, or physiognomical representation; or it may be merely of undifferentiated tetanoid character.

In the *stuporose* period or phase, catalepsy is often extreme; but the state or degree of abrogation of consciousness towards the morbid analogue of the sleep-level often appears to be a more superficial one than that which has been described under the head of typical stuporous insanity in a previous lecture.

Stadia with obstinate taciturnity and confusion of thought and of speech occur in some.

Dementia is only slowly supervenient, and rarely becomes extreme.

The order in which the stages, phases or periods occur is irregular; the circular nature of the malady is extremely irregular or abortive. One or any of them is or are apt to be entirely absent in a given case. There may be, alternately, stuporous-cataleptic phases, and excited ones; or depressed ones (as above described) and excited ones; or, successively, melancholic-depressed, stuporous, excited, confused and depressed ones; or a convulsive one, followed by apparent temporary recovery; then, successively, depressed, excited, depressed and hallucinatory, phases—in series.

There are several symptoms or symptom-groups—or connections—which mark the characteristic physiognomy of this disease clinically:—

1. The quasi-cyclical course, series or successions of differing mental states.

2. The peculiar emotional state, as shown in the pathetic voice and attitudes, in religious ecstasy, or in the emotional condition correspondent to the declamatory theatrical behaviour, yet with the impress of silliness and superficiality of feeling.

3. To the acts and demeanour just named, to the gestures repeated and of tragic or ecstatic impress, is often added a mulish resistiveness.

4. The convulsive spasmodic choreoid, or hysteroid, or epileptoid movements or grimaces, contortions, rhythmic movements, or muscular tension—the last-named especially in times of resistance—the cataleptic muscular state at some other times, chiefly in a stuporous or cataleptic phase of the malady.

5. Alternating with dumbness, the emphatic or emphasised and protracted repetition of rhymed words; of words

or phrases; or of unintelligible strings of syllables; or of the successive letters of slowly-spelled words.

These may be more special than the oratorical declamatory speech-making or recitative loquacity.

Kahlbaum indicated a tendency to use diminutives, but our language is not rich in words of the kind, and I have not observed this condition of expression in a striking form.

Case.—The following notes are incomplete, but were taken without any view to publication.

The son of a very nervous, easily agitated and deeply-moved mother, convicted of robbery, was some years in prisons; whilst there had an attack of mental disturbance with destructiveness, and later, when out on ticket of leave, got drink, was dull, depressed, and exhibited severe mental symptoms. Then during two or three weeks he sometimes stood for long spaces of time—even hours—in one position silent and immobile; or, maintaining silence, would make forward strokes with right and left arm alternately; or rhythmically jerk the head downwards and forwards, and alternately to left and right, and back again; would then suddenly stop, appear to rouse up, say a few words, and perhaps go to bed. Setting out to report himself to the police, he repeatedly retraced his steps a short part of the way. Closing a door and sitting down in the room, he would many times return to the door, open it and again shut it. The order of succession of these symptoms was not precisely made out. Taken to an infirmary by his friends he was there for several days, and was quiet; thence he came under my care, stated to be a labourer aged twenty-five, single, not suicidal, epileptic, or dangerous, and his mental attack to be of three days' duration (no doubt it was longer).

The medical certificate was to the effect that the patient would not speak or eat, remained in one position, and in whatever position he was placed, for hours; and did not understand anything said to him.

After admission he took food well until the eve of the third day when he refused tea; had an enema and small motion, and then took part of the meal.

On the morning of the third day he was again declining food, but took it later on. Viscera fairly normal; well nourished; of good muscular development, coarse limbs, dark hair, &c.; head much widest towards back, forehead narrow, but of fair height; prominent frontal eminences. Skin coarse. No external indications of syphilis.

Since admission he had been cataleptic, sitting in a chair in one position, with dilated pupils, gazing fixedly in one direction and usually more or less downwards. The only words he had uttered since admission were this morning asking once what he was "doing here."

On this day pulse 63, softish; respiration 18, very easy, and respiratory murmur very soft. The arms are kept in any raised distorted position in which they are placed by me, but the right arm not so long as the left. Rhythmical movements of the muscles of the trunk begin whilst this is being carried out. As he stands, if a leg is raised by another person it gradually and slowly sinks to the floor so that for a time the weight is slightly supported by the toes only of that foot, but after a while the heel also gets on the floor again. Under passive motion the limbs move with some stiffness rather than active resistance. When told to walk to the end of the room he does not do so, and when his arms are now replaced in their normal position and he is by gentle urging started on a walk, he only goes a step or two and then stops, until guided, gently pushed, and told to move, when again he takes a step or two. Soon he begins to tremble all over and shake as concerns his trunk and limbs (somewhat like rigor), but there is not any chattering of the teeth. At beginning of examination the eyes were directed downwards; later, they blink when fingers are snapped close to them in front, but not if snapped beside the ears, and they blink when a sudden "dab" is made in front of and close to them. The eyes are now occasionally turned by him in different directions, staring first in one direction then in another—the pupils contracting somewhat as he changes the directions. He will not put out the tongue when told to do so, and when I open his lips he immediately recloses them. These closed and somewhat pursed or pouting lips are the only part at which there is any concentration of the features. The facial expression on the whole is one slightly of sadness, depression. The face is reddish. There is no dribbling of saliva from the mouth nor any mucus running from the nostril. On the face and neck a sharp finger-nail stroke occasions a considerable effect in the form of a reddened streak of skin. He takes no notice of, and exhibits no reaction to, sharp pinches of the backs of hands or of the skin of the thighs, except as regards pupils perhaps dilating slightly and eyes looking moister. The knee-jerks are fair. No reply can be elicited—not a word is uttered by the patient. No œdema of feet. Pupils wide and sluggish.

Next day (June 8th) pulse 66. On being roused by shaking he begins to rub a cheek with the right forefinger, makes a few restless slight movements, and the face colours slightly. Markedly cataleptic.

June 9th.—Still very cataleptic, and can be moulded into all sorts of strange positions, which are retained for some time, at least, but he puts the right hand over the back and makes rhythmic flexion movements with its fingers. When I place his leg in an awkward posture he makes peculiar sounds with the mouth, inspiring as one about to endure severe pain or make effort, and continues an audible lip-sound afterwards. The knee-jerk is fair; testing the jerk is attended with greater dilatation of pupils, which are still dilated and very sluggish to light. The face is not red as it was, and its skin is very slightly desquamating. He followed the attendant into the room to me to-day, and sat down, looking at a chair. When merely told to do a thing he does not obey, but obeys at once when the order is accompanied with an appropriate gesture, and a hand is placed on him to encourage action and enforce the gesture. When he seats himself the trunk and head are well erect, and the fists rest on the thighs. If one now bows the patient's head forward it is easily put back again and even over-extended, so that the face looks up to the ceiling, the pupils also simultaneously becoming somewhat less dilated and the face colouring slightly.

During a week after this he occasionally spoke a few words, but only in reply to questions or to ask for tobacco; the limbs were cataleptic. Under an attendant he took regular exercise.

June 17th.—Addressed me; said God had told him to keep quiet; that he recollected everything that had happened; that he wanted his heart and his old woman; to get to his old woman; that I had a woman; that the attendant was "Sir;" that it did not do to "Sir" people; that the attendant had said if he did not take food a spoon would be put in his mouth, &c. He kept repeating the same sentences or parts of sentences, or slowly spelling some of the words, as w-a-y, h-e-a-r-t, d-o-c-k-r (for doctor); and repeating the words, joining others to them, with emphasis, and holding one arm forth as an orator and the other one flexed at the elbow, and its hand somewhat raised at the side. The manner and attitudes were theatrical and oratorical; the speech was declamatory; the tone of voice earnest, emphatic, pathetic. This emphatic oration of set formal fragmentary phrases and repetitions continued for nearly two hours.

June 19th.—Since the last note has spoken only in reply to questions, and to-day answers readily when interrogated, but keeps rubbing his forehead constantly and monotonously with the nail of right forefinger. The limbs are cataleptic, the hands cold and blue. Says he recollects coming here, but doesn't know *why* he was brought; knows approximately how long ago it was, and says he recollects about being at the infirmary he came from. Says that when there he "saw God and Devil, Holy God and Holy Devil, and Holy Ghost; all the world got hold of him and held him and made him kick out," and so on. Again he says he never saw a devil, then says the devil held him down and God spoke to him and told him to have trust. Says he (patient) is master, and repeats this. Will repeat the same phrase frequently, in a pathetic oratorical tone and manner, *e.g.*, "all the lot;" after repeated questions on the point he explains this to mean "all the world." Deliberately and over and over again are the phrases or sentences repeated in emphatic pathetic tone.

This condition, varied by occasional outbursts of singing and shouting, continued for some weeks, but gradually subsided. He improved, began to do a little ward-work in the middle of July, and conversed more readily and more coherently.

August 29th.—Does bed-making well, talks a little, reads a little, is obedient, wishes to go away, acknowledges he was wrong in mind and that this made him imagine he saw strange things, devils, &c., at the infirmary, but cannot give any explanation as to why he kept his limbs in any position they were placed in when cataleptic, and doesn't seem to recollect any delusion or fear or feeling of compulsion or of inertia in regard to it. Says he was in prison four years; came out in March, then was "drinking" much and taking little "food" for seven weeks, and then was improperly taken to an infirmary and brought here, although he admits he was wrong in his mind.

September 28th.—Recently had dysenteric diarrhœa. Says in one of the prisons he was at something seemed "to come over him;" he "got clouded;" he also broke up a closet, and later on "something seemed to come over him by the power of which (and not his own power) he acted;" "it seemed to make him do things." Spirits, he supposes, did it, and he seemed to hear women's voices and voices of spirits and voices of God; "to have beautiful sights at night—a lamp overhead," &c. "Really heard the voices," he says; thinks the spirits were good. Can't at all tell why he used to retain the limbs as placed in cataleptic state. Is not cataleptic now.

October 12th.—Demands release; says he never should have been sent to infirmary or here; supposes a mistake was made in sending him here; says he is had at a disadvantage, and not allowed to speak out his mind as he has a right to do; "won't stick it any longer," &c. Features twitch convulsively with emotion as he thus speaks. Has gained 9lbs. in weight since admission.

November 19th.—Looks better, works, says "he's all right." In a prolonged conversation, at first speaks of the above delussions and hallucinations at prison as being "like dreams;" subsequently speaks of them as being real, and finally admits that he still hears the voices of women talking to him and at times sees the upper part of their nude forms near him, "as if made up, or like actresses," and this by daytime, but states that he does not see them so clearly as he formerly did.

November 22nd.—Refused to wash, and became very abusive and threatening to head-attendant and others who requested him to wash.

For several weeks after this he was at times very insubordinate, refusing to wash, abusive and threatening; and declared he would wait an opportunity and attack the attendants because he was made to wash; defended this conduct, and swore lustily about his detention.

During the space of nearly a year following the date above he remained quiet as a rule, excited at times about his detention, and addressing one in a sterotyped way; stepping forward with raised arm and forefinger extended towards one's face, speaking in a pathetic tone, repeating the question, and when answered declaring he could not get an answer. In February it was noted that the patient says he used to hear a spirit speak to him in prison and see it at times, but can't say whether it was friendly or not. Also, still sees the figures or head and busts of naked women, and hears them speak, yet cannot say why they appear to him or whether they are friendly; "on the whole," don't look upon either the spirits or the women as being unfriendly. In March the symptoms last mentioned still existed, and he believed in the reality of the hallucinatory figures seen and words heard by him, but said they had ceased for the time. He alleged that he never should have been placed under care, although he went as far as to admit that the condition he had been in "was not very right, in its way." In June he got out of bed one night about 10 p.m., in an upstairs dormitory, and smashed twelve panes of glass with his fist, cutting it and losing a considerable amount of blood. This occurrence he said was the fault of those who detained him here; it was not done with any view to or possibility of escaping; and before and after it he would speak about his detention in imperfectly suppressed excitement, and in the manner already described. In September he refused food one day from mental causes. On some other days he declared he was made a laughing stock of, that this was being concealed, but that he had watched for months and knew that he was laughed at.

REFLEX SPINAL SCRATCHING MOVEMENTS IN SOME VERTEBRATES.

BY JOHN BERRY HAYCRAFT, M.D.

(Read before the Royal Society of Edinburgh.)

MANY, who have kept dogs, are aware that if the skin covering the side of the body be scratched, a dog will move the leg of that side as if itself to scratch the part touched. This fact is known to the physiologist, and a so-called, scratching centre, to which the sensory impulses are carried, and from which motor impulses to the muscles pass, has been shewn to exist in the spinal cord.

I would venture to lay before the Society one or two additional facts in this connection which have been observed by me.

First as to the sensory area from which this reflex may be initiated. The only sensitive part of the skin in most cases is that covering the lower ribs in about the middle of their course. If, however, the dog be sensitive to irritation, if it has suffered from vermin, or from any irritative condition, the area is much greater. Practically it includes those parts of the skin to which the hindfoot can be approximated. It commences posteriorly at the part which the hind leg can reach, generally two or three inches in front of the flank, varying according to the size of the animal. It extends forwards to the shoulder, including the whole side of the animal, and even reaches up the side of the neck, and on to the root of the ear. The most sensitive portion is that part which in most animals alone gives the reflex.

If in a sensitive animal the skin on the back within half an inch of the middle line be scratched the leg of that side will move. If, however, the skin at a corresponding part of the opposite side be touched, the animal will scratch at once with the other leg. The same observations apply to the scratching areas when they extend ventrally to the middle line. The skin of the flank, of the muzzle, and of the fore-leg are outside this area, and outside the reach of the hind leg. They are scratched by the teeth or fore leg of the animal. As in the case of the pithed frog, if one side of the animal be scratched, and if the leg of that side be forcibly held, scratching movements of the opposite leg may often be observed. These movements may be observed in young puppies, and can readily be called forth in animals which are sound asleep.

I have been unable to get these movements from cats, although the cat tribe is probably related to the dog tribe by common ancestry. In the rabbit, too, I have been unable to observe them. If, however, a guinea pig be killed by a blow on the back of the neck, and if the skin at the side of the belly be gently tickled the animal will bring the leg of that side rapidly to the part and scratch it violently for some time. I have noticed, too, that after an ox has been killed by a blow of the pole-axe, the hind leg will be brought to the side of the body if that part be rubbed. The movement is similar to that made during life to get rid of flies. We see then that these reflex scratchings are sometimes present, sometimes absent, in animals nearly related. This variation depends no doubt on the habits, and build of the animal itself. The cat has much more mobility of the head and neck. It can lick its sides, and can reach most parts of its body with its fore paws. The dog cannot and the hind leg is used instead. The rabbit has a mobile and flexible body, which it cleans in a sitting posture with its mouth and fore paws. The guinea-pig and the ox are shorter, with thick set necks, and the hind leg is often called into requisition.

Co-ordinated reflex movements may be divided into two classes. In the first class we have movements of limbs, the aim of which is to bring them into relationship with other parts of the body. Such are the complex movements of the pithed frog, and the scratching movements we are discussing. In these cases all that is essential for the acquisition of the power of bringing one part of the body into connection with another part is tactile sensibility of the skin. The other class of co-ordinated reflexes are those which change the position of the body in respect to its surroundings—*e.g.*, walking, swimming, &c. In this case sensation of sight, hearing, &c., are required in addition.

The nerves of tactile sensibility for the trunk and limbs pass to the cord, in which they make connection with motor fibres passing to the limbs. The nerves of hearing, sight, &c., pass to the brain.

It follows from this that the first class of movements MAY take place in a pithed animal; the latter never. Now, one constantly finds the remark that spinal, co-ordinated movements present in lower animals—*e.g.*, the frog, have their centres in higher regions of the nervous system in higher animals. This and similar remarks indicate, I think, a grave misconception.

The second class of co-ordinated movements are never purely spinal, either in the frog, or in any other vertebrate. It is probable in those cases of the first class, in which the co-operation of the brain with the spinal cord is necessary, that this is not the result merely of higher development, but depends upon other causes, some of which I have touched upon.

The scratching movements quite as complex as any of the movements of the pithed frog require for their performance the cord alone, both in the case of the guinea pig and dog. The rabbit and the cat certainly do not possess more highly developed brains; yet no such scratching movements can be elicited. The difference does not depend then upon a question of development either upwards or downwards, but rather upon a variation of habit, or build. From increased mobility of the body, or from altered habits, the cat and rabbit may have come to use their eyes and head, whereby the brain is called into action, in place of the leg used by some ancestral type. Or again it is possible that the dog and guinea pig may have acquired the use of the leg for scratching from altered habits, or from loss of mobility.

Nor is it difficult to explain the condition of things seen in the human subject. A child is born without such working connections between the sensory surfaces of its body and the corresponding groups of muscles as would lead to the approximation of a limb to any particular part of the skin. This comes only by laborious experimentation on the part of the child. It sees its foot and directs its hand to it. It feels the touch and is conscious of the movement it has made. By continual practice it can touch most parts of its body. This is learnt only by experience which has involved the use of sight, and therefore depends largely on the action of the brain.

On this account, if the spinal cord be divided we should not expect a man upon having the calf of one leg tickled to be able to scratch it with the foot of the other leg, because during his extra-uterine development the brain was a necessary factor in producing such movements. Such is, indeed, the case, for although spasmodic jerks of muscles may be called forth by stimuli applied to the skin an absence of purposive movements is noticed as soon as the cord is severed from the brain.

NUCLEAR VACUOLATION IN NERVE CELLS OF CORTEX CEREBRI.

BY JAMES R. WHITWELL,

Pathologist and Assistant Medical Officer, South Yorkshire Asylum, Sheffield.

In certain of the lower animals, and notably the Torpedo, there are large ganglionic cells in the brain, which closely resemble the motor-cells of the human spinal cord. In these the nucleolus is of large size, and stands out, in strong relief, as a highly refractile spherical body,[1] and "conceals one, or more rarely, several vacuolæ;" and this condition Max Schultze considers to be the normal form of the nerve-cell nucleolus in the torpedo. However this may be, there seems scarcely evidence that such a condition obtains in the normal nerve-cells of the human cortex.

Vacuolation of the nucleus and nucleolus of nerve-cells in the cortex, however, is present to a very marked degree in certain pathological conditions. More especially does this occur in certain cases in which dementia was a prominent feature during life, particularly the dementia resulting from epilepsy of prolonged duration.

This condition is usually most marked in the cells of the fronto-parietal region, and of these parts, the deeper layers of the cortex, especially the larger pyramidal cells in these regions, but also present in the smaller pyramidal cells, and even occurring in detached nuclei.

On examination of fresh specimens stained in aniline blue-black, after treatment with osmic acid (Bevan Lewis), and it is in specimens prepared by this method that the condition is seen best, even under the low power, it is evident that certain of the nerve-cells of these layers are deficiently stained, and in some of them the usually well-marked, deeply stained nucleus is apparently absent, or can only be seen as an irregularly shaped, more deeply stained portion of the cell. The branches of these cells are frequently noticeably deficient, both in quantity and staining; the latter perhaps being a cause of the former appearance.

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[1] Max Schultze, 'Stricker's Handbook,' Syd. Soc., vol. i.

Under a higher power, it is seen that these cells, though not deeply stained anywhere, are frequently stained in a "patchy" manner, a portion near one or other edge frequently reacting to pigment better than the rest of the cell. At the same time in many cases the sharply defined angular contour of the healthy cell is replaced by a somewhat swollen bulging appearance.

This deficiency in staining quality and somewhat swollen condition of the cell, however, appears rather to be related to the presence or absence of pigment in the cell, since this condition seems in most cases to precede the deposition of pigment in the condition known as "pigmentary" degeneration. The nucleus of the cells may present a great variety of conditions.

In almost all the nuclei in which vacuolation has commenced, or is about to commence, the sharply defined well-stained angular appearance of health is lost. In some cases the whole nucleus is swollen up to form an oval-rounded or pyriform mass, and is either as a whole badly stained, but more so at the centre than at the periphery usually, or several well-defined patches of a lighter hue appear in the swollen nucleus.

In other cases, a pale, poorly-stained, but sharply defined vacuole is detected in its centre, in the position of a nucleolus; in fact the nucleolus is swollen up and become distinctly vacuolar; in this latter condition scarcely any alteration may be evident in the body of the nucleus.

In other cases, again, in addition to the sharply defined vacuolar nucleolus, the body of the nucleus surrounding it is in a parallel condition, swollen, lightly stained, and in parts not stained at all.

In other cells, the nucleus may be occupied by several vacuoles, varying in number from 2-5; in the latter case the position of the nucleus being entirely occupied by a small bunch of these vacuoles. A still greater degree of vacuolation is that in which a signet-ring-like appearance is produced in which the position of the nucleus is occupied by one large vacuole with a small one on one side of it; in this case the small vacuole may be so compressed by the larger, as to form merely an unstained line in the capsule of the larger one.

In a few cases, again, the nucleus may be found vacuolated in one of these ways, as a detached nucleus apparently due to the destruction of the surrounding portion of the cell, which rather leads one to the conclusion that either the vacuolation itself of the nucleus, or some other condition which also produces it, is inimical to the physiological integrity of the cell. The earliest change which occurs in a nucleus about to become vacuolated is an alteration in its contour; from its normal angular form it becomes swollen, rounded, or pyriform, and coincident with the change in shape occurs an altered reaction to staining agents; instead of being deeply stained, as in the normal state, it becomes relatively lightly stained, the degree varying considerably.

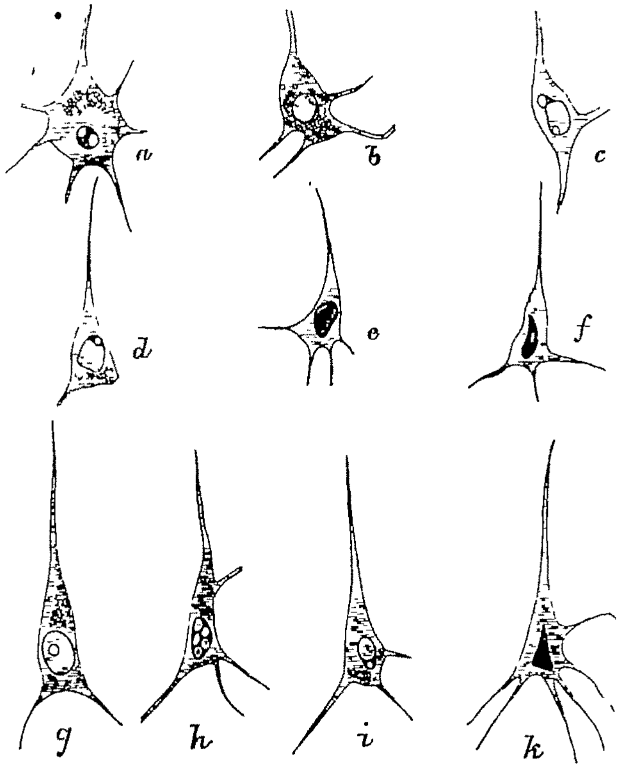
Instead, however, of the whole nucleus swelling up like this, the nucleolus alone may show these changes first, while the rest of the nucleus is only slightly altered, and perhaps bulged to one side, and this method of commencement is perhaps, on the whole, the more common.

Or, again, the condition may go on side by side in the nucleus and nucleolus at the same time.

Thus the vacuolation may commence in one of three ways, either in the nucleolus, in the body of the nucleus, or in both together.

From any one of these first stages it is readily seen that any of the previously described vacuolar conditions may be produced by a gradually diminishing staining power, and distension of the vacuoles thus formed.

The earlier conditions of the vacuolation of the nucleus are not unfrequently seen associated with pigmentary degeneration of cells, when this has advanced to a pathological degree, more especially perhaps in cases in which dementia has occurred as a sequence to a degree of mania; but pigmentation of the cell and vacuolation of the nucleus certainly occur independently of one another, since in very many cells there may be absolutely no pigmentation at all, while the vacuolar condition of the nucleus is far advanced.



EXPLANATION OF PLATE.

(*a*—*i*.) Various forms of vacuolar nucleus.

(*k*.) Healthy nerve-cell prepared by same method, from third layer of cortex, for comparison.

(*e*.) Early stage, beginning by swelling, &c., of nucleus as a whole.

(*f*.) Early stage, commencing by early vacuolation of nucleolus.

(*g*.) Early stage, process going on simultaneously in nucleus and nucleolus.

(*a*.) Large ganglionic cell from fourth layer of cortex, frontal lobe, showing "patchy" staining and vacuolar nucleus.

(*b*. *d*. *i*.) Show various forms of "signet-ring" appearance, produced by a single nucleolar vacuole at one side of a single nuclear vacuole.

(*c*.) and *h*. Show multiplicity of vacuoles.

The condition can scarcely be a post-mortem change, since it is seen best in freshly prepared sections within twenty-four hours after death. Nor can it be ascribed to any osmotic or other change associated with reagents used in the preparation of the specimens, or all specimens treated the same way at the same time would probably show the condition, which is by no means the case. In fact this form of nuclear vacuolation seems to claim a place as a distinct lesion of the nerve cell, due probably to some impaired trophic condition, perhaps the cause, perhaps the result of the dementia with which it is associated.

Vacuolation occurring in animal cells is not perhaps a very common pathological lesion, and is perhaps seen best in the forms of so-called "dropsical" degeneration, "serous" infiltration or "albuminous" infiltration.

In these forms, which occur principally in epithelial cells, the vesicular condition of the nucleus and nucleolus is frequently seen. The immediate causation of this change in these cells is considered to be due to the passive imbibition of fluid from an infiltrated tissue; but in the case of the vacuolation of the nucleus in the cortex cells, œdema is not by any means a necessarily well-marked condition, though usually present to some extent; it may be, however, that the vacuolation of the nucleus of the nerve-cell is a purely mechanical condition brought about by the imbibition of fluid, though against this is the fact, that the change very frequently commences in the nucleolus with only very slight change in the surrounding cell. That advanced nuclear vacuolation is compatible with perfect physiological activity seems scarcely possible, especially when considered with the associated changes in the cell.

[*Editorial Note.*—In order to obviate any possible question as to Dr. Whitwell's originality and priority in the results of these researches, I wish to state that both manuscript and illustrations, as here published, were in my hands as early as May, 1888.—A. DE W.]

Clinical Cases.

PARAPLEGIC RIGIDITY IN A PEDESTRIAN, ASSOCIATED WITH A REMARKABLE DEGREE OF NEURO-MUSCULAR IRRITABILITY.

BY W. B. HADDEN, M.D., F.R.C.P.

THE patient, who was a man thirty-seven years of age, came to me as an out-patient at St. Thomas's Hospital in May, 1888. When I first saw him he appeared from his gait to have ordinary spasmodic paraplegia. On testing his patellar reflexes, however, I was surprised to find that there was no increased reaction on single percussion of the tendon. I found also several of the conditions presently to be mentioned, so I formed the opinion that the affection was probably not due to gross change in the spinal cord.

The patient had four brothers all athletic men, the eldest being the ex-champion pedestrian of England. It was said that none of them were affected in any way like the patient. I was anxious to examine them but I could not get the opportunity of doing so.

The patient, though a bricklayer, had been accustomed since his youth to compete frequently in walking races. He thought nothing of walking thirty miles or more a day. Nine months before I saw him he walked to Epsom and back—a distance of thirty-four miles—in about five hours. He said that he had "never been the same man since." His gait became affected about four months before admission, and at the same time he had pain in the heels and painful cramps. The cramps were chiefly noticeable on getting up in the morning, and he used to sit on the edge of the bed until they wore off. An hour would often elapse before he could get about on his legs. The rigidity of the muscles was increased after standing any length of time and towards night it was always much aggravated. Occasionally during the day he would have painful cramps which came and went suddenly. He told me that one afternoon, a week before I saw him first, the legs became quite natural and he walked as well as ever he did.

The history therefore points to the intermittent tendency of the muscular rigidity, and the aggravating influence of exertion. Whether the affection was primarily dependent on his frequent muscular efforts is a matter which I shall defer discussing for the moment.

As regards other points in his previous history I may mention that he had a suppurating bubo twenty years previously, though there was no history of syphilitic infection. As might be expected he had been much exposed to cold and wet. A few weeks before he complained of his present symptoms he was laid up with swelling of the left knee-joint; at this time he had a discharge from the urethra, so it is very probable the attack was one of gonorrhœal rheumatism.

On admission, the patient was a well-developed muscular man, but the legs were distinctly smaller in proportion than the arms, and there was muscular weakness. The patient himself had noticed that the lower limbs had wasted. The left leg was certainly smaller than the right, but no measurements were taken at this time. At a much later period I found a difference of only a quarter of an inch between the calves, the left being the smaller and measuring thirteen inches in circumference. When the patellar tendon was struck once the knee-jerk was not over-brisk. The reactions might be termed normal. There was however a slight difference between the two sides, the left jerk being rather more obvious. On percussing the patellar tendon a few times in rapid succession the jerk became more and more marked and the quadriceps became rigidly contracted, as did also the hamstrings. This condition of tetanus or induced cramp was painful. After several attempts he would succeed in overcoming the spasm and bringing the limb back to its former condition. The same result was obtained by percussing the quadriceps itself provided that the knee was semi-flexed. In other words the muscle had to be in a state of passive tension. When the knee was extended percussion of the quadriceps produced momentary contractions which did not run into tetanus. The same condition of cramp resulted also from voluntary efforts to straighten the flexed knee against force exerted by the observer. When the limb was extended and the patella pushed down three or four times in rapid succession, as though to elicit knee-clonus, the same result followed. On pointing the toes three or four times (the method of obtaining ankle-clonus) the tibialis anticus group of muscles became rigid and stood out prominently. When the foot was left to hang down, that is when there was no passive tension of the muscles on the front of the leg, percussion caused momentary muscular contractions, not the condition of tetanus. But on placing the foot at right angles to the leg thus making passive tension, and then percussing, the tibialis anticus became rigid. When this method was adopted the calf muscles became affected, and the spasm spread rapidly to the thigh, thus producing tetanus of the whole limb. Local stimulation of the calf muscles did not give rise to rigidity, whether the ankle was flexed or extended. Both soles were very tender to pressure. When either was tickled as though to get the plantar reflex, tetanus of the quadriceps was obtained, but not of the muscles of the leg.

The peroneal nerves were not tender, but mechanical stimulation of them by the finger caused pain in their area of distribution and vigorous contractions of the muscles supplied by them, but no tetanus.

The cremasteric, abdominal, and epigastric reflexes were very brisk and slightly more marked on the right side.

The muscles of the upper limbs were very well developed though all movements were enfeebled. By the various means already mentioned cramp could be induced, but of this the patient was previously unaware. He acknowledged later, however, that his bricklayer's trowel often became rigidly fixed in his hand from cramp. When he tried to bend the arm against resistance all the muscles of the extremity became stiff, but the biceps and supinator longus more than the rest. By an effort he could extend the arm, but he could not easily bend it again. A few sharp taps on the muscles on the front or back of the forearm gave rise to local spasm of these muscular groups, rapidly succeeded by rigidity of the entire limb. As in the lower limbs passive tension of the muscles thus mechanically stimulated was an essential condition. On stimulating either ulnar nerve at the elbow by the finger, there ensued flexion of the wrist, flexion of the fingers at the phalangeal joints, and slight flexion at the metacarpo-phalangeal joints with abduction of the thumb. The thumb, ring and little fingers were affected a little before the rest. This distribution of the spasm was very brief. In a few seconds all the muscles of the limb were rigid, but the extensor spasm was predominant, and a typical claw-hand resulted. The median and musculo-spiral nerves could also be stimulated in the same manner as the ulnar, and here again the spasm rapidly became general.

On percussion, rigidity of the sterno-mastoids, the masseters, the orbicularis palpebrarum, the small muscles of the thumb, and other individual muscles could be induced, but the cramp rapidly diffused itself to neighbouring muscles.

Dr. de Watteville was kind enough to examine the patient and reported as follows: "The muscles and nerves responded rather more vigorously to both currents than in an ordinary individual. No other alteration. The course of the contraction was normal. The muscles reacted rapidly, whether stimulated directly or indirectly through the nerve, and the contraction was not protracted beyond the time of the excitation—relaxation of the muscles taking place with the usual rapidity, showing the absence of any muscular degeneration and of the condition characteristic of Thomsen's disease."

The treatment which I adopted was to keep him confined to bed, and for about three weeks he took twenty grains of bromide of potassium three times daily. Every morning he had a cold shower bath. On June 25th—just a month after admission—I made the following note: "He has been in bed all day until a week ago, when he began to get up in the evening from 5.30 to 8. The last two days he has been up from 12 noon until 8 p.m. He has been gradually improving since the treatment began. After the shower bath he says that the legs are quite supple, and he can bend them with ease. He now walks very fairly, bending the knees well. When he stands he complains of pain in the heel, and remains still for a second or two until the pain has gone, when he starts walking. All the muscle and nerve phenomena previously described persist though to a less degree. The man says he has gained four and a-half pounds since admission." He left the hospital a week later (July 1st). I noted that he walked very well, that the pain in the heels was slight, that the irritability of the muscles and nerves though present was less easily elicited, and that he had gained seven and a-half pounds since admission.

There are some other points not yet mentioned to which I must make some allusion. The man was not in the least emotional and there was no sign of anæsthesia or defect of the special senses, so often seen in hysteria. On one occasion I made an attempt to hypnotise him but without success. The man was exhibited between July and December, 1888, at the Neurological and Medical Societies, and I have to express my thanks for suggestions made by gentlemen on those occasions. In January, 1889, I again saw him and found that he was doing his work as a bricklayer. The trowel would often become rigidly fixed in his hand from cramp, but he had had no return of the permanent muscular rigidity. All the muscles and nerves as already described were morbidly irritable, in this respect there being little change from his condition on leaving the hospital. If he clenched his fist as hard as he could or shut his eyes tightly, the muscular contraction thus voluntarily induced remained for several seconds in spite of all attempts to overcome it. I could mould his hand into any shape I pleased, and the effect was to cause the parts to remain cramped for some seconds in the position artificially brought about. By means of a bright light cast by the ophthalmoscope I caused rapidly succeeding contractions of the pupils, but no permanent fixation resulted.

In this patient we see a spasmodic paraplegia, combined with a widespread irritability of the voluntary muscles and nerves which may be fitly termed "latent contracture." From the first my view was that the affection was due to over-exertion of which the history gives undoubted evidence. My opinion has lately been strengthened by a perusal of the suggestive and admirable papers which Dr. Donkin has written on this subject.[1]

It is unnecessary to reproduce here the cases cited, but it may be noticed that in all there was a history of over-exertion. One patient whose case is particularly remarkable was an acrobat, and in this instance the influence of over-use in causation and the beneficial effects of rest are well illustrated. It is not stated in the cases narrated by Dr. Donkin if there was present the extreme degree of hyper-excitability of the muscles and nerves in the upper limbs which formed such a striking feature in my patient. The evidence adduced by Dr. Donkin as to the real nature of certain cases of so-called spastic paraplegia is in my opinion irrefutable, and I cordially endorse his remark that "it is premature to treat spastic paraplegia and lateral sclerosis as synonymous; and it is certainly most unjustifiable and misleading to give the name of 'pseudo-lateral sclerosis' to cases which from their very nature seem to exclude the notion of sclerosis altogether."

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[1] "Case of Spastic Paraplegia in an Acrobat," *British Medical Journal*, December 9th, 1882. "On the Ætiology of some of the System-Diseases of the Spinal Cord," BRAIN, 1883. "A Note on Spastic Paraplegia and the Treatment of some Cases by Rest," BRAIN, 1885.

The influence of long-continued over-exertion on the functions of the spinal cord, nerves and muscles has not received the attention which it deserves, and further inquiries in this direction would probably be productive of valuable information as to conditions which are at present obscure.

Other causes in addition to over-use give rise to undue muscular irritability, and among these typhoid fever and phthisis are especially noteworthy. I confess however that I have never seen permanent rigidity occur in the course of these affections, but it is quite conceivable that certain exhausting states besides muscular exertion may cause a more or less permanent cramp which counterfeits a spinal lesion.

The condition of my patient closely resembles what is seen in hysteria, and the unusual neuro-muscular irritability is such as has often been obtained in hypnotised subjects. I am not at present concerned with the definition of that most convenient and mischievous denomination "hysteria," which it is to be hoped will some day be relegated to the category of obsolete terms. My case however is one among many which shows that definite and sufficient physical causes may lead to symptoms undistinguishable from states arising from obscure and often undetermined influences. I should be inclined to place Dr. Donkin's cases as well as my own in the category of "professional cramps," and if I had to name the condition of my patient I should call it "walkers' cramp." I do not think however it would serve any useful purpose to discuss the pathological nature of these conditions. The state of my patient may be summed up as one of permanent cramp of the lower limbs, induced by excessive muscular exertion and having no gross anatomical substratum, though identical in essential parts with the spasmodic paraplegia due to organic change in the lateral columns. I ascribe the excessive neuro-muscular irritability in the arms and elsewhere, partly to the over-use to which the body generally would be subjected in the patient's prolonged walks, and partly to a tendency of what may be conveniently termed "diffusion." In the description it will be noticed that the local stimulation of a nerve or muscle was rapidly followed by cramp of adjacent muscles and sometimes of an entire limb, and this is what I mean by "diffusion."

A case which was under the care of Dr. Bristowe at St. Thomas's Hospital about seven years ago and which I had the opportunity of observing for more than two years presented a remarkable degree of muscular irritability.[1] The history of the patient was very long and exhibited many features which, although of the greatest interest, call for no detailed notice now. About five years before she entered St. Thomas's Hospital she was attacked with symptoms of exophthalmic goitre, and rather more than three years later ophthalmoplegia externa came on. A few months afterwards it was found that she had absolute right hemianæsthesia with colour blindness of the right eye, and loss of smell and taste on the same side. Very shortly she began to suffer from true epileptic fits, and after the second of these attacks the anæsthetic right arm and leg became quite paralysed and rigid. The ophthalmoplegia externa and the right hemianæsthesia and hemiplegic rigidity continued until her death about eighteen months later. The voluntary muscles of the left (non-paralysed) side in this patient could be thrown into a state of cramp by mechanical stimulation and by prolonged voluntary effort. On pointing the foot several times in rapid succession as though to elicit ankle-clonus the tibialis anticus became rigid, the rigidity sometimes persisting for half a minute or more. The muscle also became contracted when it was stimulated by rapidly-succeeding taps. The "paradoxical contraction" could be obtained in the gastrocnemius, the peronei, the quadriceps, the flexors and extensors of the fore arm and in the biceps. The muscles of the left upper limb were thrown into a condition of cramp on the patient making a prolonged voluntary effort as, for example, clenching the fist firmly for a minute or two. The patient herself had noticed that when she went to sleep with her hand in a certain position it remained cramped for a little time after awakening, in spite of efforts to overcome the stiffness. In the notes which I made five years ago on this case the ulnar and median nerves were said not to be irritable. This patient died of bronchitis, and no change could be detected in the nervous centres after a prolonged and careful microscopical examination. In Dr. Bristowe's paper a case closely resembling the one just mentioned is described. This patient also had right hemiplegia with rigidity together with ophthalmoplegia externa from which she made a complete recovery. I believe that the phenomena of induced cramp were not obtainable on the non-paralysed side in this girl.

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[1] "Cases of Ophthalmoplegia complicated with various other Affections of the Nervous System," by John S. Bristowe, M.D., LL.D., F.R.S. BRAIN October, 1885.

It is interesting to note certain points of analogy between these cases of Dr. Bristowe's and the condition of my patient. In the woman first mentioned the rigidity of the right arm and leg and the ophthalmoplegia externa were probably instances of muscular rigidity or cramp not due to coarse disease. The extreme irritability of the muscles on the non-paralysed side and the negative result of the autopsy point to this conclusion. In further corroboration of the "functional" nature of the condition is the fact that the second patient also affected with right hemiplegia and rigidity and ophthalmoplegia externa made a complete recovery. I am quite prepared to admit that one at least of these patients, probably both, were of the "hysterical" class; but the point which I wish to emphasise is that certain nervous affections commonly ascribed to organic and coarse disease may have really no gross anatomical cause. Such conditions may depend on a well-determined cause as in my patient, or may arise without apparent reason, being then termed hysterical, as in Dr. Bristowe's cases. In the patient of Dr. Bristowe's first mentioned the permanent muscular rigidity affected the right arm and leg and the external muscles of the eyeball, whereas in my case the spasm was paraplegic, but in both cases there was excessive muscular irritability or latent contracture in the voluntary muscles elsewhere. There is therefore a close clinical and pathological resemblance between these cases, although doubtless their actual causation was different. There exist some points in common between the condition of my patient and the rigidity which occurs in Thomsen's disease. In the latter however the spasm occurs when the muscles are put into action after a period of rest and rapidly disappears with the continuance of exertion. The rigidity never tends to assume permanency as in the cases which I have given, as well as in Dr. Donkin's cases. With rare exceptions Thomsen's disease is congenital and hereditary, and arises usually at an early period of life. Moreover the electrical reactions in my patient, as Dr. de Watteville observes, are different from what is found in Thomsen's disease.

Finally I would call attention to the beneficial effects of treatment by rest in my case—a point which also has been strongly insisted on by Dr. Donkin.

Critical Digests.

ON PARALYSING VERTIGO (GERLIER'S DISEASE.)

BY P. LADAME, M.D. (GENEVA).

Translated from the Original Manuscript by E. B. Shuldham, M.A., M.D. Oxon.

DURING the summer of 1885 Dr. Gerlier, of Fernex, had observed a few cases of this curious complaint, which he first brought under the notice of the Medical Society of Geneva on November 3rd, 1886. Its tendency to spread, like an epidemic, into the neighbouring villages, and to disappear with the onset of cold weather was then noticed. A few cases were noticed in July, 1886, and another outbreak occurred in August, 1887.

In his first communication[1] he points out as three pathognomonic signs—(1) muscular resolution, (2) cervical pain, (3) ocular disturbances. These symptoms occur in a more or less intense, frequent and regular mode, and occur sometimes daily like ague attacks, for periods of ten minutes at most, between which the patient appears to be enjoying perfect health.

Symptomatology.—Muscular resolution may affect any portion of the voluntary motor system, but chiefly the extensors and the left side of the body. It is bilateral, though not usually symmetrical. One leg is weaker than the other, for instance; and in the case of a shepherd one hand was still capable of performing the act of milking, whilst the other remained helpless.

The author says, "I thought at first that the shepherd suffered from cramp, analogous in character to writer's cramp, which Basedow has described by the name of 'milker's cramp,' while on second thoughts I believe that we have to deal with a muscular paralysis—in fact a paralysis which makes the workmen drop their tools; and in the shepherd's case the same thing takes place.

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[1] *Revue Médicale de la Suisse Romande*, Jan., 1887.

"Besides, one day I saw one of my patients during an attack with his hand crooked up and unable to raise the three last fingers. The extensors were paralysed. I took his hand and raised these very fingers without any difficulty, but they fell back to a state of flexion as soon as I let go of them."

M. Gerlier has also at times noticed in some exceptional cases a kind of paralytic stammering. One of the patients complained, amongst other matters, of losing control over his tongue, and of not being able to roll his food about in his mouth during mastication. The medical man at Fernex also speaks of a case of paroxysmal dysphagia caused by paresis of the pharyngeal muscles. As soon as deglutition began it was with great difficulty that food passed the isthmus of the gullet. However, I will here remark that the picture of symptoms of paralysing vertigo is exceedingly variable. There is nothing so changeable as the appearance of a patient during a fresh attack.

M. Gerlier has already marked out three distinct types of the affection: one where the patient appears to be blind drunk, one where he is in a state of contemplation, and one where he seems to be standing asleep; without taking count of all the other varieties.

Incomplete Observations.

The author reminds us that we must not lay too great stress on the observations which he has made of his cases. He says, "My notes do not represent a series of consecutive observations made at leisure, but they are rather scraps of information seized as the occasion permitted; often furnished and noted down in the open air, *sub tegmine fagi.*" We must admit that M. Gerlier has turned to excellent account these scraps of information which he looks upon as incomplete. He has described paralysing vertigo with a master hand in his arrangement of the scattered material which he has brought together. He has succeeded in creating a remarkable type of this affection, but unfortunately it is not a fixed type, so that the reader loses his bearings just as he is on the point of getting the true definition of the disease in question.

Pain in the Head inconstant in Character.

Next in order to the muscular paresis comes pain in the head as a pathognomonic symptom of paralysing vertigo. But the uncertainty of this symptom is even, if possible, greater than the preceding one. After having told us that pain in the back of the head with stiffness of the neck is a constant sign of this kind of vertigo, M. Gerlier also says that the pain in the head can be exchanged for a pain in the loins, so that a spurious lumbago follows a spurious torticollis. These painful spots become centres for other pains to radiate and flash out from, which resemble the lightning pains of posterior myelitis, but differ from them by being less severe and by lasting longer. It may even happen that the patient finds chief fault with the radiating pains, and overlooks the spinal cord from which they originate; that is to say, the patient has neither a spurious torticollis nor a spurious lumbago, but a pain over the left eye.

The eye troubles make the third pathognomonic symptom of the affection which is the subject of my remarks. However, ptosis is at present the only objective symptom met with in examination of the eyes.

The author himself recognises the fact that the falling of the eyelid belongs to symptoms of muscular paresis. He says, "If I give this symptom a place apart to itself, it is because this is the most constant sign of this proteiform disease, the least variable and the easiest of verification. The eye troubles then are essentially subjective in character —cloudiness of sight at the beginning of an attack, double vision, sparks, oscillation, dancing of objects before the eyes."

M. Gerlier moreover remarks that the eye has an undoubted influence in causing an attack. The bustle of human beings in towns, the flowing of water under bridges, the glare of strong light, the gazing at any large open space is enough to bring on an attack of vertigo. You see we have to deal with a psychic impression, whose source is in the sight.

Insufficiency of the Characteristics of the Vertigo.

If now we try to find out the true nature of this vertigo, which appears to us so important that it has been given a fresh name, the author tells us that it has nothing to do with dizziness, though the people at Collex, who believe it results from drinking absinthe, have given it the expressive name of (tourniquet) turnstile.

An attack can be produced at will by making the patient look steadfastly at some bright object. He then complains of a vague feeling of malaise, soon followed by ptosis but not by nausea, as is the case in gastric vertigo. On the other hand Dr. Gerlier says: "Nothing brings on an attack more easily than for the patient to look into space. I often make use of this influence to make sure of my diagnosis and at least to cause ptosis. The physician can believe in such a symptom as 'dread of the market place' (agorophobie) if by chance he witnesses this very symptom produced by the patient looking at any public thoroughfare and is unable to ask him any questions." We regret that the author has not devoted a further study to this symptom. He only speaks of it in reference to the eye troubles as a kind of accessory symptom, but gives it no special notice in any of his writings.

In the year 1887 M. Gerlier saw some fresh cases of this paralysing vertigo towards the end of the summer. For the first time he published nine observations—a *résumé* of which is here given (*v. Revue Médicale de la Suisse Romande*, Nos. 1 and 2, pp. 22 and 86).

1. *Five cases of an Epidemic which broke out at the Farm at Ornex.*

Observation 1.—On the 7th October, 1886, nine months before the appearance of the actual epidemic, the shepherd at the *Château d'Ornex* complained of the following symptoms:—

T. Z., fifty-two years of age, unmarried; has been suffering for some days past from attacks which were chiefly characterised by sudden pains in the middle of the back extending to the nape of the neck and causing a feeling of being strangled, mist before the eyes, falling of the eyelid, especially of the left eye, which is completely hidden; the head can no longer be raised, but falls forward on the chest, the back is curved; the patient motionless and staggering on his legs, is obliged to wait till the end of the attack, which lasts for one or two minutes.

The act of milking causes numbing of the shepherd's hand and paralysis of the fingers, pain in the nape of the neck and disturbance of sight comes on at the same time. Often, however, this kind of paralysis occurs without the presence of any concomitant symptom. This attack comes on all the more frequently if the shepherd has a great number of cows to milk, which makes his task a very painful one. The frequency of the attacks varies from day to day, but as a rule when they have well commenced they are brought on in easy succession. This patient relates that having ridden in a vehicle on the previous evening, his attacks followed one another uninterruptedly whenever he looked straight before him. During the whole of the winter and spring of 1887, T. Z. has been free from his complaint.

It was only during the first few days in July that he began to feel any difficulty in milking the cows in the afternoon. The left hand was the first to be paralysed, and he was soon obliged to leave off his work for some minutes. At one time there was simply inability to milk the cows, at another he suffered also from weakness of the neck, and disturbance of the sight.

On the 13th of October, M. Gerlier reported the following symptoms:—Ptosis more marked in the left than in the right. Neither mydriasis nor myosis, nor irregularity of the pupil, nor strabismus was apparent. The patient did not suffer from rotatory vertigo during the attack, but rather from dulness of vision, at times from double vision, and even from sparks before the eyes. It is true that his attack was accompanied with pain over the left eye and heaviness of the head, symptoms not usually met with in paralysing vertigo. If I made him raise his head, and look even at the cloudy sky, he became dizzy and felt that an attack was impending—at the same time his ptosis became worse. Reading had become an impossibility, so he was obliged to give up his daily paper; as soon as he looked closely at the print everything was blurred, and, besides, his eyelid fell, and his frontal muscles vainly contracted in the effort to raise it again. Matters were even worse, it seems, when he tried to read by lamplight. He also suffered from trembling of the hands which is very marked when the arm is extended. The trembling is severe enough to prevent the patient from writing, though he manages to sign his name, but his signature is like that of a man suffering from general paralysis. There is no embarrassment in the speech, no paresis of the tongue, and no difficulty of swallowing. His general state is good. He is a tall, muscular man, and has lost neither sleep nor appetite. He complains of perspiring easily, and of a heaviness in the head in the afternoon. He denies any excess in drink, but, on the other hand, has got into the habit of taking little sips all day. Rapid improvement followed the use of cold baths and better ventilation of his stable.

Observation 2.—D., thirty-nine years of age, married the father of three children. Hired as an agricultural labourer at the Chateau farm in the spring of 1887, he worked uninterruptedly to the 10th of August. His first indisposition dated from the end of June, but the attacks really appeared in the last fortnight of July, during the harvest. The attacks came on every day in more or less frequent succession, from about three to eight o'clock in the evening. The patient describes his sensations:—"All at once my sight was affected, and I could not see ten paces in front of me. I was obliged to rest on my spade and to stop quiet for a moment. I felt something on my back like a band, and my head inclined strongly forwards without being able to raise it. The pain spread to my loins and my legs. I felt paralysed and unable to raise my feet."

The patient's appetite and sleep was excellent. Neither heaviness of head nor profuse perspiration.

Observation 3.—P. I., unmarried, thirty years of age, robust in health and of good constitution. Sleeps at the farm in a room by himself. He is head servant. Some light attacks before July 31st, when he attended the Federal Rifle meeting at Geneva, and was very much upset all day long. He was quite unable to raise his eyes on the plain at Plainpalais or to look at distant objects. The attack did not begin with disturbed vision nor with the rotatory vertigo, nutation, nor with sparks before the eyes, but with simple dulness of vision. A cloudiness comes over his eyes and he is unable to see things distinctly. At the same time the eyelids fall, and the eyes turn to the left. Paresis of the neck muscles; the head falls on the chest, but there is no spurious torticollis, nor pain in the spinal cord. Weakness of the legs does not come on in all the attacks. Trembling of the lips..

Observation 4.—M. A., forty-one years of age, married, father of three children; farm bailiff. For some time past his labourers have complained of attacks characterised by disturbed vision and inability to raise their heads, while he had never suffered from anything of the kind. On Thursday, July 28th, he went in a *char-à-banc* to the Federal Rifle match, and arrived at Geneva about eight o'clock in the morning, when his sight was clouded and his eyelids drooped. When he came on the ground and found that he could not look at anything without disturbed vision, he took refuge in the canteen, where he remained all day. About five o'clock in the evening he ventured to walk, but was immediately attacked by feeling as if there was a bar across his neck, and then his head fell completely forward on his chest. For some days after he had a repetition of the attacks in the afternoon, but less severe. He was cured at the end of August. In other respects his general health was very good. Sleep and appetite unimpaired. After the cure of his attacks a slight dulness of vision remained.

During the second hay-making M. Gerlier noted two attacks of quite a peculiar character. The patient suddenly felt a weakness of the trunk muscles on the left side, so that his body was bent on one side, and he would have fallen on his shoulder but for the support of his hay-fork. Then an absurd symptom occurred; he had a feeling of emptiness at the waist, and thought that if he could only laugh he would be cured.

Observation 5.—L. T., day labourer, thirty-nine years of age, married; father of three children. Steady, sober man, smokes, but not to excess. Has been working at the farm uninterruptedly since spring. Towards the end of June he began to suffer from attacks which he attributed to the thin sour wine he had been drinking with the rest of his fellow labourers. He said, "All of a sudden my sight was disturbed, my eyes closed as though I were going to sleep. I had a feeling of a cord being passed round my neck, and I was unable to raise my head. I then placed my hand before my eyes as if awaiting sleep for some minutes." He went to the Federal Rifle Match without being inconvenienced, but his brother who worked at the same farm, and who was with him at Geneva, could not walk, and had the look of a drunken man.

Besides these five persons, three others were also affected in the same house, which raises the total of patients to eight out of twelve people who lived in the farm of the Château d'Ornex. The neatherd, the carter, the farm bailiff, and the serving maid were the only persons who escaped the affection.

M. Gerlier adds: "I have also learned that a shepherd employed on the farm during the month of October was in his turn attacked during seed-time whilst the others were cured, or on the point of getting well. In fact, the cases that appeared during this epidemic were not simultaneous in character, but came in succession. The shepherd dates his relapse from the first week of July; the two labourers (Observations 2 and 5) were attacked towards the end of June, the head-servant towards the 15th of July, and the farm bailiff on the 26th of July. This is quite enough to establish the fact that the food had nothing to do with causing the attacks, even setting aside the proof of the immunity enjoyed by four people. However, the cause of the mischief was certainly at this very farm, for I did not meet with a single case of a like kind in the village of Ornex, and the epidemic was absolutely limited to the members of this household. I found another proof in the fact that the four day-labourers who were living at Ornex, and who left the farm before the 10th of August, suffered no more after this date, while on the other hand the shepherd and the head-servant (Observations 1 and 3) had the most serious attacks at the end of August.

Observation 6.—T. R., nineteen years of age, and of good constitution, drinks no absinthe, neither smokes nor chews nor commits excess of any kind. Is not at all overworked. Some disturbances of sight during his attacks from the beginning of June. In August his condition was worse. At the end of August the attacks were accompanied with a painful sensation of strangling—the pains radiating all along the spine, and making the patient bend his head forwards. There was no rotatory vertigo, but a simple mistiness before the eyes, which chiefly came on when he wanted to look at distant objects and which left off as soon as he looked for a moment on the ground. Very often double vision was present, also ptosis, but neither mydriasis, myosis, nor irregularity of the pupil. Muscular paresis not strongly marked, but very varied. Trembling of the lips, but not of the hand. Inability to milk, but after dinner he takes a *siesta* in an unhealthy, badly-ventilated stable.

Dr. Haltenhoff, who saw him on the 1st of September, reports that there was no dulness of vision, but rather defect of accommodation. Reading was out of the question.

No discomfort after the 30th of September. On that day he came to Geneva to be inspected and to pass the examination for being received as a military recruit. He got along very well all the morning, but in the afternoon he could not write nor go through the tests, on account of his disturbed sight. These were his last symptoms.

Observation 7.—P. M., eighteen years of age; was seized with the attacks suddenly. He could not see clearly, and complained of a feeling of weight on the nape of the neck.

Observation 8.—Z., thirty-two years of age, bachelor, was affected at Bossy the previous year, and had some idea that he was suffering from "le tourniquet." First attack on the 11th of August, and since then every afternoon an attack. One day when he was holding the plough handle he let go of it during an attack and fell on the ground. On Sunday the 10th of September he had a drinking bout. The following day he could not go to work; on Tuesday he returned to work, but during the morning he had four attacks which caused him to fall to the ground. The attacks are varied, but offer the characteristic symptoms. Pains in the nape of the neck extending all along the back, down to the ankles and the feet, and always accompanied by paresis of the leg muscles. The eye troubles consisted of simple amblyopia at first, soon followed by diplopia and then by rotatory vertigo and nodding vertigo. Z. has seen the trees, as it were, falling, and the plough swaying to and fro before him. These are exceptional phenomena of paralysing vertigo. Very different kinds of paresis affecting the extensors of the head, the trunk and lower limbs.

Observation 9.—F. M., strong girl, twenty years of age, housemaid, rises before daybreak between 3 and 4 o'clock in the morning to milk the cows. She then takes the labourers their breakfast in the fields. She puts her hand to everything, and can handle the spade at need. The attacks display the three classic symptoms: pains in the nape of the neck, troubled vision with ptosis, bending of the head forwards on the breast, occasionally paresis of the lower limbs. This girl has never suffered from paresis in milking the cows, which the author attributes to the slight fatigue caused by her having to milk only three cows and the recent date of her illness.

She is the only patient in the house. The stable is unhealthy and badly ventilated. M. Gerlier saw this girl two or three weeks after in the fields, working in the same line with two labourers who were digging potatos.

However, we cannot help expressing our deep regret that there is such an absolute lack of information in the personal and hereditary antecedents of the patients, their habits and previous mode of life, their psychic mould—all matters of infinite importance when we have to deal with interpreting nervous or mental phenomena. For it is noteworthy that a whole set of symptoms, such as we have just described, is capable of very different interpretation according to the point of view which we choose to take.

Before going any further with this discussion we should reproduce in brief two papers of MM. David and Haltenhoff which treat the same subject in a slightly different manner, and bring their precious contingent of personal observations, which are very often independent of those which have been made in the neighbourhood of Fernex-Voltaire.

The epidemics described by M. David are of singular importance, and we think that his observations will throw more than a mere ray of light on the nature of this strange affection.

The first epidemic observed by M. David broke out in the farm of Château de Collex in 1884, that is to say, a year before the first of the Fernex cases. Now, the Château de Collex is a few minutes' walk from Ornex, and the distance from Fernex is about two kilomètres. These are facts of great importance in the matter in hand. We confine ourselves to the bare mention of them for the time being.

In the epidemic which broke out at Collex in September, 1884, M. David observed nine cases of illness among the twenty persons who made up the population of the farm. They are as follows:—

(1) M., shepherd, thirty years old. He came on the estate in the spring. During the attack of ptosis he lets his head hang languidly on his shoulders, and has the gait of a drunken man.

(2) A. L., servant, aged twenty; has been in service on the estate for four years. He was affected during the summer of 1883 and 1884. No attacks during the autumn.

(3) C. G., shepherd, aged twenty-three. He came in the spring and left the farm in June, owing to the attacks, which greatly alarmed him. He was free from them in his new situation. Returned in July as field labourer, but was immediately seized by a fresh attack, and so was forced to leave once more.

(4) B., twenty years of age, employed to look after the pigs; came in the spring. The same symptoms are observable in his case.

(5) L. E., aged thirty-six, from Bossy; not an abstainer. He came on July 3rd, and suffered from the attacks which he had never had before.

(6) D., not an abstainer; the same attacks.

(7) Mme. X., the cook on the farm, mother of a family; came on the estate on September 1st. The first attack came on on September 15th. There was no defective vision; slight diplopia; then came weakness in the nape of the neck and weakness in the legs—particularly the right leg. There was no ptosis, and she continued her work.

(8) Louis C., aged twenty-seven; applied to Dr. David on September 27th, 1884. He is a tall, strongly-built man, having all the appearance of good health. His appearance is very singular. He looks as if he could not help falling asleep; his eyelids are fallen, and he cannot raise them, except with his fingers; no irregularity of pupils. There is no strabismus; the patient, however, avers that he sees double. He carries his head straight, but with difficulty. The tongue is not furred.

He came on the farm at Collex on June 1st, in perfect health; three weeks after his arrival he noticed that he saw double, and that he was unable to open his eyes fully A little later, while at work, he was seized with fits of weakness, attacking in turn the nape of the neck, then the arms and legs; he felt himself grow heavier and heavier, and could no longer hold his tools. His head fell forward, *his stomach was contracted.* This state lasted some minutes;

the attack then diminished in force, leaving ptosis and diplopia.

(It was after having observed this patient that M. David went to the farm to see the others, of whom Louis C. had spoken.)

(9) L. M. applied for advice on October 7th, a few days after the above-mentioned visit.

L. M. is thirty-nine years of age, and is not an abstainer. He alleges that at Villars-Dame, in July, 1884, he had a previous attack, consisting of dizziness of the head and paralysis of the neck. He came to Collex on September 22nd, and had no attacks there. He left the next day for La Belothe, where he had a severe seizure while gathering nuts. His head fell forward. In alarm he returned to Collex. The malady increased, and he could not hold his head up; his arms, hands and legs became paralysed, and he had a sensation of dragging in the stomach, and he fell down at every tenth step he took. As soon as he lies down the attack ceases; he alleges that he had to lie down ten times on the road from Collex to Versoix. He says that he is in this condition the whole day long; as soon as he tries to work the attack begins. Only a slight degree of ptosis is observable in his case. Sent to the provincial hospital; he left it after a few days without having had any attacks.

Cases in 1885.

In the following year, 1885, there were two fresh cases at Collex:

(10) S. Jacob, aged fifty-nine, father of a family, for fifteen years carter on the farm of Collex, a moderate drinker, has suffered for a year (in summer, and during the daytime only) from a feeling of heat in his eyes, which become hazy; then there is an indefinite sort of pain passing from the nape of the neck to the forehead over the crown of the head; the head begins to tremble, the legs and body become weak, and unless he lies down he falls to the ground. As soon as he lies down the symptoms abate. There is about ten minutes' interval between the time of his first feeling the approach of the attack and the moment of his falling. The attacks recur

several times during the day. He believes them to be due to the sunlight; if he covers his eyes or avoids the light the attack passes off. This patient succumbed on the 4th of May, 1886, to an attack of hemiplegia on the right side.

(11) A. L., fifty-four years of age, proprietor at Collex, has suffered from attacks of sudden weakness during work, since October, 1885. He cannot raise his head and feels a heaviness of the arms and of the whole body. This weariness lasts all day in more or less marked degree; he has no vertigo, nor eye symptoms. He has never felt anything like this before.

Epidemic of Genthod in 1886.—The second epidemic noted by M. David occurred at Genthod (a short distance from Collex, near the border of the Lake of Geneva), at the farm of M. M. The outbreak dates solely from the year 1886. The author gives the following account of it:—

"The first cases appeared on the 20th of June, 1886. At the end of a few days the number of patients was at its maximum. Out of seven men who worked together there were six attacked. The attacks always took place during work in the great heat of the afternoon. They were characterised by troubled vision, accompanied by heaviness of the head and a feeling of weakness, which spread from the nape of the neck, to the neck, the arms and the legs. No pain was complained of. These different symptoms appeared in succession and with increasing severity as soon as the patient stooped, and ceased as soon as he rose up again. The farmer who was working one day about seven o'clock, and who together with a man of fifty-one years of age, was the only one exempt from the vertigo, gave me a dramatic description of the appearance of these six men making superhuman efforts to pick up a few blades of wheat, and when the cart was loaded sinking down exhausted at the end of the field as though they had just accomplished some extraordinary task. He laid particular stress on the curious appearance of these labourers, with their half-closed eyes. He thought at first that they were amusing themselves, but he soon saw, to make use of one of his own expressions, that they were obliged to resort to their fingers to raise their eyelids. All these men of different ages, but chiefly young men, were attacked for the first time, with the exception of one only who stated that he had suffered from the same thing two years ago at Frontenex.

"After the heavy work of harvest was over there were no more cases. The vintage, the ploughing, and the sowing, passed off without any fresh attacks."

With reference to the different characters of the two epidemics, M. David makes the following remark: "It is needless to insist," he says, "on the great care with which we must accept the statements of people who are often bad observers, and with whom the imaginative and the imitative faculty play a part which must never be lost sight of in sifting their statements. It is probably in these elements by which we must note the differences which exist between the two local epidemics, of which I have been the part observer."

According to my honoured colleague at Versoix, the affection is characterised in brief by a vertigo which is accompanied by general weakness, which begins in the muscles of the eyelids and extends successively to the muscles of the nape of the neck, the arms, and finally the legs. There is little or no pain; sensation is intact. One of the patients said that he could perfectly feel the flies which lighted on him. No formication and no numbness.

Finally M. David concludes by saying, "Heavy work in the great heat of the sun is without doubt one of the determining causes of the attacks. Exposure to the sun would not of itself explain this kind of vertigo, which would ere this have certainly been noted amongst the numberless cases of sunstroke due to military exercises, where all sorts and conditions of men run this particular risk. Auto-suggestion, and above all imitation, with more or less interested motives attached, might be suspected in one or two cases, but this cannot explain the great majority of them. In conclusion, I must admit that great heat and strong light during heavy work determine the attacks, but I am unable to explain their exact and intimate cause."

Dr. David's observations already show that paralysing in perfect harmony with the classic observations of MM. David and Gerlier. To us it seems clear that in our choice of observations we must proceed with greater accuracy if we want to give the term "paralysing vertigo" its earlier meaning; unless we do so we should soon allow all the cases of toxic symptoms due to ingesta to be included in the term and many others besides.

We shall see that the abuse of alcohol and venereal excess also play a great part in the etiology of Gerlier's disease.

Observation 4.—F. C., farm labourer at Varembé, (August 25th, 1875). Has been suffering for the last month from pains in the nape of the neck, which radiate to the sides of the neck and sometimes to the throat, where he feels a constriction and difficulty in swallowing. Heaviness of the upper lids, which sometimes are closed involuntarily. Incomplete double ptosis. He attributes his symptoms to being exposed to draughts. M. Haltenhoff learned that F. C. is a drunkard, and that he is the only one attacked on the farm.

Observation 5.—A. V., twenty-seven years old, field labourer at Percy (August 12th, 1878.) In the preceding month of June he was suddenly affected with ptosis during the hay making. Daily attacks at four o'clock in the afternoon. Dull pain in the forehead. General health very good. Vision normal. *The patient admits to some excess in drinking white wine.* He attributes his symptoms to the action of sunlight during work.

Observation 6.—L. G., twenty-six years of age, vine grower, at Chongny, May 2nd, 1884. For the last month has been suffering from heaviness of the eyelids, chiefly in the afternoon. In the evening feels stiffness in moving the lower lip. Quite recently general muscular weakness in the neck, the arms and the legs; no excesses (?); no signs of syphilis; no paresis beyond that of the levator muscles of the lids. He came for advice about his double ptosis. He is the only patient affected in this way. It appears that two years before his attack a labourer on the same farm had shown a set of analogous symptoms.

Observation 7.—E. P., twenty-nine years of age, head footman (Oct. 26th, 1886.) Was seized some three weeks before this date with a severe pain in the nape of the neck and weakness of the legs, which made him fall if he leaned forwards. Sudden visual disturbance with frontal headache. No ptosis. Nothing revealed by the ophthalmoscope. The farmer attributes the defective health of his employé to *constant sexual excess.* He always had a haggard look about the eyes, and some time before this had shown signs of the same nerve weakness.

As for the following observation, we may possibly have to deal with a case of neurasthenia or of dilatation of the stomach, for it is quite impossible to know exactly the nature of the case from the information given; but it certainly does not belong to the class of "paralysing vertigo," as can be seen by the description which follows.

Observation 8, Sept. 24, 1886.—E. D., thirty-six years of age, field labourer at Collex, married, father of a healthy child, temperate in his habits, always in good health, was seized in June with pains in the nape of the neck, giddiness, pains in the head, worse in stooping, which affected him all through the summer. At times double vision, cramps in the hands and arms, muscular weakness of the right leg with slight loss of sensation. In the month of August troubled vision, severe and persistent pains in the head between the eyes, difficulty in opening the eyes, especially when in the sunlight. He sleeps well and is very impressionable; a sharp sound makes him tremble. Has a weary look about him, and an expression of suffering. Earthy complexion. Pressure on the occiput and the cervical apophyses, which are tender, causes a feeling of shivering in the shoulders and arms. Excess of phosphates in the urine. Tendinous reactions very exaggerated. Formication in the limbs. Eruption of urticaria, possibly a quinine rash. Sent for medical treatment under Professor Revilliod; he leaves the hospital cured in a few days. He comes under M. Haltenhoff's care on June 11th, 1887. Although he says he is cured, yet he remains excitable and is easily tired. Every morning on rising his left hand is numb and tingles. He suffers from difficulty of swallowing and even of mastication. It appears that his four fellow-labourers have been attacked by symptoms of paralysis. The shepherd could no longer milk the cows; the carter slept in the stable. At the same time there were some cases at the Château de Collex farm. The labourers were all obliged to leave the farm after the second hay making, because they were unable to work. The patient D. declared that this state of affairs had been going on for the last three years.

The Importance of Psychic Influence unrecognised.

The last assertion made by the patient enables one to grasp one of the most frequent causes of the spread of the paralysing vertigo as an epidemic. Although M. Haltenhoff and M. Gerlier are both agreed in rejecting the possibility of psychic contagion, yet to explain the etiology of these epidemics, we cannot for a moment question the importance of this psychic influence, as we shall soon be able to show.

The last case noted, No. 9, by M. Haltenhoff is that of Jean Reber, of Colovrex, sent by M. Gerlier to have his eyes examined, with the result that they were found to be perfectly normal. M. Haltenhoff, however, found that the patient could not read, and that he was suffering from traces of ptosis of the lids. Jean Reber was one of M. Gerlier's first ten cases, which formed part of the little epidemic which broke out during the summer at the farm of Colovrex (a hamlet which belongs to the district of Fernex, Collex and Genthod), and which attacked four out of seven labourers—the shepherd Jean Reber, the neatherd, the carter, and the female servant. M. Gerlier, who gives an account of this epidemic in his work 'On the Origin of Paralysing Vertigo' (p. 262), says that Jean Reber showed symptoms of paralysing vertigo in a very marked degree. In his case not only was ptosis, false torticollis, and paresis of the neck muscles to be seen, but also the patient was unable to milk, to mow or to wheel his barrow, and temporary paralysis of the lower limbs, with falling to the ground, was present.

Discussion at the Medical Society of Geneva.

Such are the facts. Now let us examine the various ways in which the said facts have been interpreted. Since the reading of the first paper at the Medical Society of Geneva by the physician of Ferney, on November 3rd, 1886, the discussion which was raised has shown a great divergence of opinions. M. Revilliod saw in hospital one of the cases which had been sent by M. Gerlier. At the time of the patient's entrance into hospital he showed no very marked symptoms; he complained of a little weakness of the legs, but what he chiefly dreaded was a return of the first symptoms of his disorder. This man chewed tobacco, and at first M. Revilliod thought that he could attribute the symptoms present to the toxic influence of the tobacco, but a close examination, together with M. Gerlier's report, did not allow him to admit this hypothesis. M. Picot had Jean Reber under treatment at Pneuré. The patient chiefly complained of inability to milk the cows. M. Picot was led to think of an affection analagous to writers' cramp, which might be called shepherds' cramp. M. Martin saw a case where the patient attributed his symptoms to the wine which had been given him to drink

M. Goetz sees an analogy between the affection just described and gastric vertigo, but certainly the two affections are by no means identical. The influence of large quantities of different drinks taken by the field labourers during summer cannot be slight.

M. d'Espine does not think that one can attribute the cause of this epidemic to ptomaines; in fact, the patients have not shown any disorder either of bowels or of circulatory tract.

The discussion was renewed in the Society's rooms on December 1st, 1886, where we heard Dr. David read his paper. M. d'Espine communicated the following observation.

It refers to a man thirty years of age who came to consult him on Nov. 22nd, 1886, for an attack of herpes zoster. This man, who was a field labourer, had been working at a farm during the summer at Maconnex, near Ferney, and it was there that he had suffered from attacks of giddiness some three or four months previously; the attacks showed all the characteristics observed by M. Gerlier. The giddiness disappeared as soon as the shingles came out. This man showed no trace of alcoholism; he took his meals with his superiors, who were exempt from the affection; he drank no wine but only cider, and he seemed to have no knowledge of the fact that there were others besides himself in the neighbourhood of Ferney and Collex who had symptoms like his own. In brief, what chiefly struck M. d'Espine in this case (besides well-marked anæmia from which he suffered) was the regularity with which the attacks of giddiness returned every afternoon, whilst during the morning the patient was quite free from them.

M. Revilliod gave an account of two patients who have shown the same symptoms and had been under his care at the hospital. One of the patients had not shown any of the symptoms characteristic of ptosic vertigo (this was the first title which M. Gerlier gave his paper) since his reception into the hospital. Although he had been given some fatiguing work to do, M. Revilliod thought that the cause of these symptoms should be looked for in the alimentary canal.

M. Hilt, judging from the regular recurrence of the attacks of vertigo under the usually happy influence of change of residence and of sulphate of quinine, and from the information given by M. David, thinks that one of the causes is possibly malaria. M. d'Espine sides with M. Hilt in this aspect of the case.

Finally, paralysing vertigo was the subject of a third debate at one of the meetings of the Medical Society, when M. Gerlier made his communication on the origin of this affection on April 6th, 1887. It is now time to give the theories developed by the physician of Ferney, so as to explain the cause and progress of this new affection.

The Cowshed Microbe.

According to M. Gerlier one can compare the invading progress of ptosic vertigo to that of mildew in the neighbourhood of Geneva. During the summer of 1885 mildew was found in some vine trellises and gardens; in July, 1886, it attacked all the vineyards. In his first paper he says: "I am fully disposed to view this new epidemic as the result of toxic symptoms caused by microscopic cryptogams. The appearance of the vertigo in the month of July and the cessation of all fresh cases in October is a very abnormal occurrence, pathologically considered. It coincides with the mushroom season and with the ravages of mildew."

In another paper M. Gerlier comes to the conclusion that the miasm of paralysing vertigo (the existence of this miasm has never been discussed by him) takes up its quarters in the cowshed, and that the infecting influence escapes from the cowshed just as the tetanus is supposed to escape from the stable.

This is why more cases were met with in the practice of Drs. David and Gerlier than in that of their colleagues, whose practice lay at the foot of the mountains. The neighbourhood of Geneva, the necessity of supplying the town with milk, made the neighbouring proprietors keep their cows in summer. "In our district a large suburb of Geneva," says the author, "the cowsheds are full all the year round, consequently their sanitary condition is not so good as those at a greater distance from the town, and the effluvia arising from them are more penetrating." However, there were some who escaped infection although they were exposed like the rest to the miasmatic influence. But this fact does not embarrass M. Gerlier. Are there not some individuals who do not take measles, scarlet fever, or yellow fever? It appears that we can acquire immunity from paralysing vertigo by having had a previous attack, or by constant exposure to the miasm, although all the cases which have hitherto been seen prove the contrary. Till now, this miasmic origin of the affection is but a mental aspect of the case, and not the result of any direct observation.

We here quote M. Gerlier:

"1. This disorder takes an epidemic form in the house, not because it is contagious, but because the focus of infection is indoors.

"2. It chiefly attacks the farmer and the agricultural labourer, because they are in special contact with the sheds.

"3. It attacks the shepherd and the farm servants first, because they frequent the stable more than all the rest of the household.

"4. The small grower is more often attacked than the man who farms in a large way, or the rich landlord, for he is obliged to look after the cattle himself, and to take the shepherd's place.

"5. The strange immunity enjoyed by women is due to their having so little to do with the stable.

"6. Lastly, there is no room for surprise if change of residence has a favourable influence on the affection and even often checks its progress, because it removes the patient from the source of infection."

M. Gerlier concludes by saying, "All the facts therefore agree in supporting this view of the case—the microbe is caught in his lair, and the origin of the infectious matter can be traced to the sheds."

What certainly causes me surprise is that such a distinguished *savant* as our honoured colleague of Fernex should find these facts sufficient to sustain his arguments in favour of "the cow-shed microbe theory."

M. Gerlier adds, "The miasm of paralysing vertigo bears the greatest analogy to that which causes the black vomit; cold destroys it. But it has found in our climate, with its severe winters, a locality where it can hibernate, a hothouse fit for its existence; he has found the cowshed," &c.

Mental Contagion.

In the meantime, as these interesting researches have commenced, it might be fairly asked whether the epidemics of paralysing vertigo could not be partly explained in another way? Since M. Gerlier's first communication saw the light, we have been struck with many circumstances described by him, which have made us think that possibly we had a kind of mental epidemic to deal with in the spread of this remarkable neurosis.

The progress of this disorder, the terror which it inspired in the country districts, its progressive invasion of villages which lay close to each other, the superstitious centre where it was to be found, its sudden and complete disappearance as soon as the patients changed their residence, all these circumstances made us suspect some psychic influence in the outbreak of these epidemics. It stands to reason that we made this supposition unreservedly and solely with the view of drawing the attention of our honoured colleague of Fernex to the possibility of an explanation of this kind, which has not occurred to him. M. Gerlier was obliged so show that this supposition was inadmissible, and M. Haltenhoff undertook a formal refutation of the same. We will pass in review the arguments which have been brought forward by our honourable colleagues against the possibility of a psychic contagion, and we will then proceed to discuss the weight of their arguments. This will be a singularly easy task, on account of the statements which have just been made.

In his paper of 1886, M. Gerlier said that he met with ptosic vertigo in seven villages and hamlets on the Franco-Swiss frontier, Collex, Fernex, Magny, Colovrex, Saconnex-le-Grand, Ornex, Maconnex, and he adds these astonishing words:—"These villages, though close together, have no intercourse, and the patients are unknown to one another."

This statement appears incomprehensible to us. We have visited the above named villages. They are united by fine wide roads, and the most friendly relations appear to exist between the inhabitants. The roads are incessantly traversed by pedestrians, carriages and carts, and we cannot possibly make out how the inhabitants of neighbouring villages, whose very fields lie close together, could be so isolated as to avoid contact with each other. However, M. Gerlier who lives in the district, undoubtedly knows better than we do what goes on there, and we must admit as true his statement that the patients were not acquainted

The Magny patient told us afterwards of his own free will that they attributed this affection to witchcraft. He told us, without giving us his own views on the case, that many patients went to consult the sorcerer at Plan-les-Outes, near Geneva, because they were convinced that such a strange affection could only be caused by the "Evil Eye," or sortilegè.

M. Gerlier confirms this statement. He tells us the following: "Nevertheless, it is reported that there is a suspicion of the shepherd's being in a weak mental condition. I am convinced that he is so on account of his strange behaviour. At the farm he plays a part of his own. He is a kind of specialist who has nothing in common with the other servants; he does not do field work, but he is constantly with his cows in the sheds; his ideas are limited. The shepherd is superstitious and believes in witchcraft. He is uncleanly and very careless of his person." He never was married, despises women, and shows affection only for the cattle he has raised. But on the other hand he is entirely unemotional, and anything but a prey to nervosism." This may be so, but he is superstitious, and therein lies our reason for suspecting that patients affected with the disease, and held to be victims of the "evil eye," propagate the disease by moral contagion among those who are still further predisposed to infection by depressing conditions, such as over-fatigue, insolation, alcoholism, &c.

These considerations further explain the fact of *female immunity* from the disease, inasmuch as they show women to escape the predisposing influences to the contagion. Dr. Gerlier himself says: "A peasant, and his son, who cultivated their own fields at Maconnex, fell a prey to the disease. The wife, who had escaped, told me with a laugh that 'women were never taken ill.'" It is to be noted however that women who work out of doors with fi labourers in times of an epidemic may be affected als shown by two cases recorded by Drs. David and G and we may expect that as soon as women beco scious of the possibility of personal infection, become subject to the disease as much as the m present immunity, therefore, is by no means absolute, and cannot serve as grounds upon which to base any theory.[1]

To sum up: Our view is that the cases which fairly come under the title of this critical review constitute a group with the characteristic features of an epidemic. We reject the hypothesis of an organic infection in favour of that of a cortical disturbance, in the spreading of which psychical contagion acts as an important factor.

[illegible] cases of the disease, according to Drs. Gerlier, David and Mestral [illegible] in 1888, unless we admit as accurate a report to the effect that some [illegible] Château farm were showing symptoms so analogous to those ob[illegible]n, as to have given apparent support to the theory of a common [illegible]rin.

Reviews and Notices of Books.

Inebriety, its Etiology, Pathology, Treatment and Jurisprudence. By NORMAN KERR, M.D., F.L.S., &c. Second Edition. H. K. Lewis, 1889.

IN spite of a celebrated series of articles on the "Comparative Study of Drunkenness," which perhaps first opened the eyes of the medical world to the wealth of material that is contained in the phenomena of drunkenness, the study of this subject has by no means attracted the attention or the industry that it deserves. The student who wished to refer to an accurate description of a single case of the degrees of mania and of dementia produced by the immediate action of alcohol, would not know where to lay his hand upon the record. And yet the importance of the subject is very great, for by means of alcohol we can artificially produce insanity in the sanest individual. We can induce—or rather the subject himself, for our edification, will induce in himself—by rapid or slow degrees, a gradually progressive insanity, ending at last, as all insanities do if pushed far enough, in coma. The value of this experimental aid to the study of insanity is manifestly enormous, and it is a matter of surprise and of regret that hitherto the opportunity thus given us has been so little utilised. When, therefore, we find a work avowedly devoted to the study of inebriety, we open it with perhaps an exaggerated anticipation of the value of the material that it is likely to contain. It appears, however, that by inebriety Dr. Norman Kerr does not mean drunkenness. Inebriety is, according to his definition, not the conditic
being drunk, but the condition of desiring drink. This d
this craving—or "crave," as he prefers to call it—is,
Norman Kerr's eyes, in itself a disease—is in fact the
to whose study his book is devoted. Inebriety is, h
diseased condition, "the characteristic symptom o
an overpowering impulse to indulge in intoxication
In another place he defines it somewhat differen

stitutional disease of the nervous system, characterised by a very strong morbid impulse to, or crave for, intoxication."

This position is a very definite one, and from his point of view, inebriety and drunkenness are very different things. "The act of drunkenness," says Dr. Kerr, "is no more the disease of inebriety than is a violent deed the disease of insanity. There may be intoxication and there may be murder, without any apparent underlying disease. But no inconsiderable proportion of drunken eccentricities and of fatal criminal assaults are the product of diseased states. As there is a morbid condition designated 'insanity,' so there is a morbid condition designated 'inebriety.'"

In bringing forward a proposition of this character, and in seeking to erect into a condition of definite disease a state of things which has hitherto been looked upon as wholly or largely a vice or moral delinquency, the onus of proof lies upon Dr. Kerr. It is for him to establish the position by such evidence as he is able to produce. But in producing his evidence he is by no means as clear nor as free from ambiguity as in stating the proposition that he wishes to prove. Of direct evidence, indeed, as distinguished from positive assertion, he brings forward but little. Again and again we are assured that inebriety is a disease, but beyond a statement of the Archbishop of Armagh's, the proof that he adduces is extremely feeble. In stating the relationship, which he says is very close, of inebriety to insanity, he falls into a curious mistake. He endeavours to prove the closeness of the relationship by pointing out how closely the conduct in drunkenness resembles the conduct in insanity. But surely this, according to his own showing, has nothing to do with the case. If we admit, as I for one am rejoiced to admit, that drunkenness and insanity are not merely alike, but identical in their manifestations, we do not on that account admit that *inebriety*, as defined by Dr. Kerr, is allied to insanity. We prove, indeed, that drunkenness is allied to madness, but "inebriety" is not drunkenness. It is "a disease characterised by an overpowering impulse to indulge in intoxication at all [illegible]" and what Dr. Kerr should prove is that this disease, [illegible] that drunkenness, is allied to insanity.

[illegible] impression left upon the mind by this portion of the book [illegible] Dr. Kerr somewhat stretches the received meaning of [illegible] he speaks of a morbid impulse as a disease. By [illegible] commonly meant an alteration of structure, and this [illegible] recognised by Dr. Kerr in another section, in which he speaks of the pathological appearances of inebriety. Under this heading are detailed the changes in the mucous membrane of the stomach, the nutmeg liver, the fatty heart, the granular kidney, &c. But these are the pathological appearances not of inebriety, not of the impulse to drink, but of the effects of drink. If inebriety is to be considered a disease apart from drunkenness, then its pathological appearances and alliances must not be confounded with those of drunkenness. This confusion of the two states that have avowedly been separated prevails throughout the whole book, and vitiates the greater part of the reasoning for scientific purposes.

When we turn from the scientific to the practical part of the work, we find evidences of greater care. Dr. Kerr has evidently had great experience in the study and treatment of drunkenness as well as of inebriety, and he gives the results of his experience with candour and with evident desire for accuracy. He has, however, become so deeply impressed with the evil effects of drunkenness, that his hatred for alcohol in all its forms, and indeed for stimulants generally, amounts almost to fanaticism. He is eager to accept upon evidence that to a less biassed mind appears insufficient, any and every evil story that can be told about alcohol, and attributes to it effects which may well have resulted from other causes. It is doubtful whether the cause of temperance, or rather of abstinence, that he so earnestly and enthusiastically cherishes, is not injured rather than served by this indiscriminate advocacy.

When Dr. Kerr attributes habitual drunkenness to the casual imbibition of a glass and a-half of champagne at a Christmas dinner, or of a glass or two of port or sherry, he is drawing too deeply upon his belief in the villany of his enemy. If such dread consequences follow from such trifling causes, and if the habits of taking opium, chloral, chlorodyne, ether, eau-de-cologne, ginger, capsicum, gelseminum, sumbul lavender, and other drugs can be acquired in the same way, ought we not logically to give up the use of drugs altogether? Dr. Kerr himself advocates the administration of gentian, calumba, cascarilla and bark, but how can he feel any confidence that in doing so he is not laying the foundation in his patient of some horrible "crave"—of gentianism, calumbomania, cascarilliasis, or barkism?

All that Dr. Kerr says about the effect of habitual excess in alcohol in destroying the love of truth, and even the perception of truth, is undoubtedly in accordance with fact, but it seems as if an opponent—an *advocatus diaboli*—could retort by adducing the effect of habitual abstinence in distorting the sober judgment, and producing a tendency to exaggeration, not of fact, but of inference.

CHARLES MERCIER.

Traité Pratique des Maladies Mentales. Par le Dr. A. CULLERRE. Small 8vo, pp. 618. Paris, 1889.

THIS excellent manual, from the pen of the medical director of the Roche-sur-Yon Lunatic Asylum, is a concise treatise on psychological medicine as taught by the French school of to-day.

Its contents are arranged under four heads: *Introductory*, comprising a short historical survey of mental medicine, considerations relating to the pathology and pathogeny of insanity, and the classification of its various forms; *Part I.*, devoted to questions on general pathology; *Part II.*, containing the description of individual forms of mental alienation; *Part III.*, in which the medico-legal relationships of the insane are considered.

The first subject calling for notice is the classification—a subject which early attracts one's attention as being, in some measure, an index to the author's originality and his command of the matter treated. In this case, after critically examining many of the nosologies hitherto constructed, the author elects to base his system upon the French classical model, introducing such addition and modifications as are necessary to bring it into harmony with the existing state of medico-psychological science in France.

In tabular form Dr. Cullerre's system stands thus :—

- I. Primary Alienations
  - 1. Simple Insanity
    - Mania
    - Melancholia
    - Periodic Insanity
      - Intermittent Insanity
      - Insanity of double form
      - Circular Insanity
    - Progressive systematised delirium
  - 2. Degenerative Insanity
    - Hereditary Insanity
      - Mental *obessions* ; irresistible impulses
      - Systematised delirium of hereditary cases
      - Reasoning mania, moral insanity
      - Polymorphic deliria
  - 3. Idiocy

II. Alienations associated with organic cerebral affections	General paralysis (diffuse interstitial encephalitis) Senile dementia (cerebral atheroma with consecutive atrophy) Organic dementia (focal lesions).

III. Alienations associated with various general morbid conditions	1. with Neuroses	Epilepsy Hysteria Chorea
	2. with Poisons	Alcoholism Saturnism Morphinism Pellagra Cretinism

A few condensed quotations from the author's expositive remarks will assist in making plain those portions of the classification which are not immediately obvious.

"There exists a first category of alienations which are not accompanied by any fixed and determinate lesion of the nerve centres. It is divided into two groups, in the first of which the mental troubles are uncomplicated by degeneration; until the onset of the malady the brain has functioned normally, the individual has been accounted sane. But in the second group anomalies of mental function preceded the outbreak of confirmed insanity. From the first, or from an early age, mental instability, morbid impulses, or perversions of character or intellect have existed. This vast group of psychic troubles only develops in defective brains stamped in diverse degree by degeneracy, congenital or acquired. In the great majority of these cases that degeneration is congenital and must be regarded as the result of accumulated neuropathic heredity, whence the expression *hereditary insanity*.

"The form *dementia* is discarded on the ground of its being only a secondary condition, a mode of termination of insanity, properly speaking, and no longer recognised as a special form of mental disease.

"The group *monomaniæ*, still retained by Marcé in 1862 but rejected by his contemporaries, is replaced by *hereditary insanity*, which, for simple disconnected psychological entities, substitutes the notion of a permanent affection of the nervous centres having very diverse manifestations according to the individual circumstances, but connected the one to the other by the continuous bond of the degeneracy."

The above nosology does not differ substantially from the one recently submitted to the Medico-Psychological Association of Paris by a commission of members of that body for adoption as the ground-work of an international classification. It is easy to note defects in it, as in all others; nevertheless in certain particulars it contains evidence of a decided advance upon the systems to be found in most, if not all, English text-books. We refer especially to the recognition and definition of that assemblage and sequence of symptoms designated *progressive systematised delirium* as constituting a special form of insanity, and the comprehensive grouping effected under the term *hereditary insanity*, the first being a development elaborated in the lowest order of facts subservient to classification—semeiotic; the second a progression (imperfect, it is true) to a higher grade—etiological. In both these matters, however, there are many exceptionable points involved.

Those readers who have not followed the progress of mental pathology in the hands of modern French alienists may perhaps find some interest in a fuller reference to the results just mentioned. We, therefore, purpose to extract from Dr. Cullerre's description such brief outlines as may serve to indicate the chief features of the innovations.

"Under the name *progressive systematised delirium*[1] we describe a form of insanity of chronic evolution and slow course, characterised successively by uneasiness, suspicion, painful hallucinations of hearing and general sensibility, and by systematised delusions of variable nature, leading most often to the transformation of the personality, to megalomania, and finally to dementia.

"For long confounded with melancholia (Pinel) or with monomania (Esquirol) this form of insanity began to be isolated by Lasègue in 1852, who described the chief phase of it as being a special form and type of mental alienation which he denominated *delirium of persecution*. That new teaching was gradually accepted by the generality of physicians.

"Thanks to the labours of J. Falret, Magnan, and their pupils, it has been recognised that the delirium of persecution is only a syndrome pertaining to a more complex malady. Considering that the principal characters of this type of alienation are the systematisation of the delusional ideas, and the progressive evolution of the delirium, we have designated it as above. The nomenclature proposed by Magnan, *chronic delirium*, is too vague; that used by Garnier, *progressive systematic psychosis*, or the one suggested by Ballet, *chronic progressive systematic psychosis*, might be adopted.

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[1] Dr. Cullerre's definition of delirium aptly illustrates the meaning attached to the word by French writers: he says, "The *ensemble* of the delusions constitutes the *delirium*; their origin, their connection, their nature, their number, give to the delirium its form and its differential characters."

"The etiology of this affection usually is obscure. The common moral causes of insanity occasionally count as determining agents. Amongst those whose action appears to be less hypothetical must be placed a reverse at law, and the fact of being an illegitimate offspring; this latter circumstance, by placing the individual in perpetual conflict with social prejudices and interests, creates in him an exceptional moral situation, which is very liable to act perniciously upon his intellect. Deformities, particularly of the sexual organs, sometimes have a similar influence.

"Heredity plays a more considerable *rôle* than is indicated in statistics (36 in 134 cases of this disease at Charenton), but in most of the cases it is simple heredity—not complicated by degeneration. In patients in whom the hereditary influence is marked by congenital mental instability, the course and symptomatology of the progressive systematised delirium present characters which reveal its origin. The frequency of this affection is considerable. Lasègue placed it at 14 per cent., A. Foville at 19 per cent. The recent researches of Planès seem to demonstrate that those proportions are too high. Of 32,000 insane persons who passed under observation from 1872 to 1885, 8 per cent. belonged to this category; the percentage, however, increased annually during the latter portion of that period. The female sex is much the more prone to the malady, the ratio being 5 to 3.

"The age at which the disease develops is comprised between thirty and fifty years. Most of the patients are widowed or celibates.

"Four stages of the disease may be noted: (1) *The incubative period*, during which the patient is gradually invaded by a vague sense of inquietude, of mental suffering, indefinable *malaise*, giving rise at first to astonishment; then distrust awakens, and soon the patient is suspicious of all around; by degrees he forms the suspicion that he is persecuted; trifling, commonplace, independent events nurse in him the idea that they are signs contrived to annoy him. But so far his delusions are indistinct. The development of auditory hallucinations marks the termination of this stage.

"(2) *Delirium of persecution.*—The patient's belief in the existence of persecutors is only secondary; it is evoked by the need he feels for some explanation of his morbid experiences, and which he only succeeds in satisfying after the appearance of the hallucinations of hearing. At the commencement there are elementary hallucinations—noises, whispers, blowing sounds, &c.; subsequently they simulate actual voices, appearing to issue from the floor, walls, &c. Their tenour invariably is offensive.

"Thus far the systemisation of the delirium is rudimentary; the patient believes that he is persecuted by enemies, but he does not define who or what they are. The systemisation, however, progresses either slowly by psychological deductions, or suddenly and spontaneously. According to the patient's education and environment he attributes his persecution to witchcraft, electricity, &c., actuated by one or more persons.

"As the disease advances hallucinations of other senses develop, and the more these troubles become complicated and multiplied, the more complete becomes the systemisation of the delirium.

"The order of frequency and mode of association of the sensorial disorders are as follows:—

"(1) Hallucinations of hearing.

"(2) Hallucinations of hearing and of general sensation.

"(3) Hallucinations of hearing and of general sensation; also of smell and taste.

"(4) Hallucinations of hearing; also of smell and taste.

"Visual illusions may be present, but hallucinatory disorder of this sense is always absent—this fact constituting one of the distinctive characters of the disease. Should visual hallucinations be found present, they indicate some complication—more especially alcoholism.

"The troubles of general sensation are multiform. Morbid sensations referred to the sexual organs are of very frequent occurrence—almost constantly present in females.

"The hallucinations of taste and smell always are associated with the notion of being poisoned, and give rise to most varied delusions, often with reciprocal conduct.

"The auditory hallucinations become greatly extended in range and complexity; their nature also undergoes change; from being *auditory verbal* hallucinations they become purely psychic. After having held conversations *in thought* with their enemies, the patients end by being strangers to the functioning of their own intellect; their ideation becomes automatic; they then imagine that their thoughts are stolen from them; that their ideas are divined before they be formulated. Thus, little by little, a sort of doubling of their personality is brought about: they seem to be possessed by another individual who seizes their ideas and utters them from without or from various parts of the body.

One patient described this state as "exile"; she said she was exiled from herself.

"When it has attained its definitive systemisation the delirium is attended by remarkable peculiarities of language. Each patient invents a special vocabulary to express his delusions. Not content with creating new expressions, they modify words and the ordinary written characters, transpose, suppress, or alter certain syllables, change the letters, &c.

"*Delirium of grandeur.*—The evolution of grandiose delusions indicates the transition into this stage. These delusions seem to develop either by logical deduction or by spontaneous genesis. To illustrate the former method—a patient, after brooding over the unjust ceaseless persecution to which he believes he is subjected, arrives at the suspicion, and finally at the conviction, that he is an exceptional being of exalted rank and power, that he is persecuted by those whose interest lies in excluding him from his rights. The second method is displayed by those patients who, without any premonitory evidence of change, suddenly one day proclaim some extravagant delusional conception.[1]

"*Dissolution of the delirium, Dementia.*—In this last phase of the disease the systemisation of the delirium breaks down; the morbid conceptions become lost in a medley of confused and disconnected ideas of persecution and grandeur, the expression of which becomes more and more incoherent and incomprehensible.

"COURSE—DURATION—TERMINATION.

"The onset of the disease is in most cases insidious and slow. The period of incubation may extend over many years. The second stage, especially, may be extremely long; twenty or more years may elapse before the third phase be reached. On the other hand, in some cases, principally those marked by hereditary degeneracy, the malady runs a rapid course, the various syndromes follow with great rapidity, or may develop simultaneously, and the disease attains its full development in a few weeks. Besides irregularities in the duration of the various stages, the occurrence of the stages themselves is far from being constant. Some patients never pass beyond the first period, and, according to J. Falret, only 34 per cent., and Mairet, 42 per cent., reach the third stage.

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[1] This mode of development was faithfully exemplified a few days ago in a case under the reviewer's notice. The patient, A. R., a married woman with hallucinations of all the senses excepting vision, for fifteen years has suffered imaginary persecution from her former neighbours: "They inject brimstone, ether, and everything that can make people sleep: they inject opium and chloroform in at the door and down the chimneys; they inject *mawks* to cause vomiting." When at home she wore a tea-tray as a breastplate "to keep the stuff from going into" her chest. In the asylum she habitually covers her mouth, ears and nostrils to prevent ingress of the noxious substances. On the day in question, her usual demeanour of calm endurance suddenly changed to one of elation, and she declared that she was about to be married that morning, her titles were Queen Victoria, Lady Champness, &c. This striking mental alienation was attended by marked physical change—pallor and increased debility.

"Remissions are common. The disease usually is incurable, and ends in dementia. The recovery percentage (5) recorded by one observer is too high, through the inclusion of incorrect diagnoses and incomplete recoveries. Occasionally the disease merges into delirious mania, or melancholia with stupor, but this manner of termination is rare, and only supervenes in non-typical cases.

"This affection requires to be carefully distinguished from melancholia with ideas of persecution. In the latter there is general depression of the faculties, a state of anguish; the melancholia is self-accusatory, or self-condemnatory. On the contrary, the sufferer from progressive systematised delirium, preserves a certain equanimity, and attributes his troubles exclusively to external agents. The tremor and short duration of the delirium in subacute alcoholism, together with persistent insomnia, dyspepsia, the mobile character of the delusions of persecution, and the presence of visual hallucinations, serve to differentiate it.

"In one category of hereditary cases styled *persécutés raisonnants* that might be confounded with progressive systematised delirium, the delirium is never completely systematised; it changes its object and form, and is not associated with hallucinations of general sensibility; it does not progress towards megalomania or dementia, and the persecutions complained of belong purely to the sentimental order.

"In another class of hereditary insanity, the delirium may assume the form of progressive systematised delirium in its second and third periods. The differential diagnosis here rests upon the antecedents of the patient, the sudden outbreak of the delirium, its imperfect systemisation, and its habitual association with maniacal excitement or melancholic depression. The distinction is important, because this pseudo-attack very often is curable.

we find *onomatomania*, a form described by Charcot an occurring in persons high in the scale of degenerate a word or name plays the chief part. This disorder diverse types: (1) the agonising search for a name or wo the urgent obtrusion of a word and an irresistible imp repeat it; (3) the particularly fatal significance of words; (4) the preservative influence of certain words the word seems to be a veritable solid body, swallo weighing upon the stomach and capable of being ejected by e pulsive efforts. In all these cases the patients retain entir consciousness of their state; they deplore their absurd ideas, bu are none the less slaves to them.

The polymorphic deliria are chiefly the vesaniæ of Class I. Division I., occurring in hereditary subjects.

With few exceptions Dr. Cullerre's book throughout is writte in an easy lucid style. The clinical features of the severa maladies are delineated with the force of a skilful observer an able writer.

We do not meet with anything novel under treatment; th principles enunciated are thoroughly sound, and the details o therapeutics recommended are equally good, save that in th description of means to evacuate the stomach in cases of acut alcoholism the author has overlooked the stomach pump. Here an there we note that Dr. Cullerre has not divorced himself from th too prevalent custom of enumerating certain drugs which hav been advanced at various times, but which in truth are utterl useless in the cases mentioned.

The sections on pathology do not add fresh material t current views and knowledge. The merits of the book, however far outweigh such defects as exist. Dr. Cullerre has admirabl succeeded in the project to which he applied himself.

E. BIRT.

END OF VOL. XII.

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"HEREDITARY INSANITY.

editary predisposition dominates the whole etiology of alienation, but its action is not the same in all cases. typical forms of insanity previously considered (Class I., n I.), that predisposition usually is only disclosed by the k of insanity itself. The patient, prior to becoming insane, ibited nothing abnormal in the manifestations of his psychical ctivities, and on recovering from the attack he returns to a normal mental state. But in the group of psychopathies now in question, the heredity is revealed from the commencement of psychical existence, and its action is apparent throughout the life of the individual; the functioning of the mental faculties always deviates in greater or lesser degree from the normal, showing unequal development and want of harmony. This defect of proportion between the several faculties of mind forms a basis from which mental troubles—exceedingly numerous and variable—incessantly spring; and these are the two morbid elements—the one fixed, the other mobile—which precisely constitute that which should be understood by the term *hereditary insanity*."

The defective points in this nomenclature are discussed with much fairness by Dr. Cullerre. It is inexact, because applied to only a portion of hereditary cases, and because it includes cases in which the degeneracy is not of ancestral origin, but owes its existence to some agent, such as cranial injury or one of the specific fevers. Clinically, there are no fixed limitations; many simply ill-balanced minds, belonging to the hereditary class, do not present other signs of degeneracy. Physical evidence of degeneration may exist without symptoms of hereditary insanity occurring; one may find amongst typical maniacs or melancholics individuals who display pronounced marks of degeneracy.

The mental and physical stigmata of the insane diathesis are well described. Dr. Cullerre divides the subjects of this heritage into four classes—the intelligent, weak-minded, imbeciles, and idiots.

Mental *obsessions* (a word which admits of no adequate universal rendering, and is one of the many instances in which the superiority of the French psychologists' vocabulary is shown), or the state in which the mind is possessed by some irrational and progressive fear, such as agoraphobia, alone or conjoined with an irresistible impulse, are the first syndromes considered in the subdivisions of hereditary insanity. Amongst these obsessions